DOES NOT CIRCULATE

BRITISH MEDICAL JOURNAL

JOURNAL OF THE BRITISH MEDICAL ASSOCIATION

UNIVERSITY OF MICHIGAN
JAN 8 1953
MEDICAL LIBRARY

SATURDAY JANUARY 10 1953

TABLE OF CONTENTS

Epidemiology of Some Skin Infections ROBERT CRUICKSHANK, M.D., F.R.C.P., D.P.H. 55
Triethylene Melamine in Human Malignant Disease
EDITH PATERSON, M.B., F.R.C.P.Ed., P. B. KUNKLER, M.B., M.R.C.P., and A. L. WALPOLE, Ph.D. 59
Tuberculosis Case-finding by Tuberculin-testing of Infant-school Children
I. A. MACDOUGALL, M.R.C.S., L.R.C.P., D.P.H., J. R. MIKHAIL, M.R.C.S., L.R.C.P., and W. H. TATTERSALL, M.D. 64
The Acute Chest in General Practice JOHN FRY, M.B., F.R.C.S. 68
Dissecting Aortic Aneurysm: A Varied Clinical Picture R. K. MACCUISH, M.B., M.R.C.P. 71
Leaking Abdominal Aneurysms J. WOODROW BETTS, F.R.C.S., and B. C. ROWLANDS, F.R.C.S. 73
Food Supply, Body Weight, and Activity in Great Britain, 1943-9
J. M. HARRIES, B.A., and DOROTHY F. HOLLINGSWORTH, B.SC. 75
Haemolytic Anaemia Treated by A.C.T.H., Cortisone, and Splenectomy
J. D. AITCHISON, M.B., F.R.F.P.S., M.R.C.P. 78
Diffuse Phlebosclerosis with Calcification A. CHARAN, M.S. 80

MEDICAL MEMORANDUM
Neonatal Death due to Ligation of Umbilical Cord by an Amniotic Band. F. O'K. CRAVEN, M.D., and C. J. M. GEDDES, M.B., Ch.B. 81

REFRESHER COURSE
Head Injuries—II. D. W. C. NORTHFIELD, M.S., F.R.C.S. 90

GENERAL ARTICLES AND NEWS
Medical New Year Honours 92
NOVA ET VETERA 93
Infusion Pump. V. J. KEATING, M.B., D.A. 94
UNIVERSITIES AND COLLEGES 108
MEDICAL NEWS 113
BIRTHS, MARRIAGES, AND DEATHS 114
QUESTIONS AND COMMENTS 115

OBITUARY
J. Cole Marshall, M.D., F.R.C.S. 104
Gibbon FitzGibbon, M.D., M.Ch., F.R.C.P.I., F.R.C.O.G. 104
H. R. Nutt, M.D., F.R.C.S. 105
Sir T. Drummond Shiels, M.C., M.B. 106
Arthur Cant, M.B. 106
F. S. Cliff, M.D., M.R.C.P. 106
Thomas Mackenzie, M.D. 107
G. T. Watson, O.B.E., F.R.C.S. 107
E. J. Foulds, M.B. 107
H. E. Rutherfoord, M.D. 107
E. H. Hunt, M.Ch., F.R.C.S. 108

Vital Statistics 111

LEADING ARTICLES
Autonomic Control 84
Scientific Study of Delinquency 85
Radiology on Points 86

ANNOTATIONS
Outbreaks of Skin Diseases 87
Medical Care of the School-child 87
The Toxicity of Iron 88
Soft-tissue Radiology 89
The New Year Honours 89

REVIEWS
Oestrogens and Neoplasia 82
Actinomycosis in General Practice 82
Antibiotics 82
Infant Development 83
Medical Statistics 83
Poets' Infirmities 83

CORRESPONDENCE
Pulmonary Moniliasis. L. P. GARROD, M.D. 95
The Neurology of John Hunter's Last Illness. Sir WILLIAM COATES, F.R.C.S. ..95
Modified P.A.S. in Genito-urinary Tuberculosis. J. G. GOW, F.R.C.S.Ed. 95
Intra-arterial Injection of Tubocurarine and Thiopentone. J. N. FELL, M.B. ..95

SUPPLEMENT
Full Contents on First Page of Supplement
General Medical Services Committee 7
Remuneration of Hospital Medical Staff ..9

CORRESPONDENCE—*cont.*
The Backward Child. D. J. SALFIELD, M.D. 96
Flogging. C. G. LEAROYD, M.R.C.S. 96
Physical Medicine. J. W. PAULLEY, M.D. 96
Iron Therapy. R. S. SAXTON, M.B. 97
Christmas Disease. D. H. COLLINS, M.D.; P. R. KEMP, M.B. 97
The College of General Practitioners. J. K. PATERSON, M.B.; BERNARD KENDRICK, M.B.; A. G. CRUIKSHANK, F.R.C.P.Ed. ..97
Advertising in Examinations? L. M. FRANKLIN, M.R.C.S. 98
Anaphylaxis after Penicillin. ARTHUR BEAUCHAMP, M.B. 98
Self-demand Feeding. C. WILFRED VINING, M.D. 99
Period of Gestation. A. L. GUNN, M.D. ..99
The Catheter and the Prostate. WILSON HEY, F.R.C.S.; CHARLES WELLS, F.R.C.S. 99
Mind and Skin. H. GAINSBOROUGH, M.D. 100
The Patient Tells His Story. G. GRANT, M.B., and R. A. N. HITCHENS, M.B. 100
Automatic Dispenser. E. JAMES, M.D. ..101
Dermatomyositis and Malignant Disease. E. W. PROSSER THOMAS, M.D. 101
Intussusception Associated with Trauma. A. A. MURRAY, F.R.C.S.Ed. 101
Thrombocytopenic Purpura in Successive Pregnancies. J. A. CHALMERS, M.D. ..102
Radio-iodine and Thyroid Carcinoma. E. E. POCHIN, M.D. 102
Ear Syringing. J. K. M. DEAS, M.B. 102
Sarah Gamp's Pharmacopoeia. J. A. ROSS, M.D., F.R.C.S.Ed. 102

BRITISH MEDICAL ASSOCIATION, TAVISTOCK SQUARE, LONDON, W.C.1

No. 4801, WEEKLY, PRICE 3/-

Copyright

REGISTERED AS A NEWSPAPER

pure soap
no perfume
no colouring
no fillings
that's

The Lily—the symbol of Purity

SIMPLE SOAP

The definition of "Purity" is:—
"Complete freedom from mixture of any kind". This faithfully describes "Simple" Soap which is of natural colour, and guaranteed to contain no Aniline Dyes, Perfumes, or Fillings such as Titanium—not even animal fat—as these, Dermatologists and scientists have established, are often the causes of bad skins, setting up or aggravating Dermatitis. Many Dermatologists and doctors are recommending "Simple" Soap to patients whose skins are allergic to ordinary soaps, and as a non-medicated cleansing agent for troublesome skin conditions.

"Simple" Soap makes no claims to medication—its purity is its only claim to distinction.

Made by the makers of the World famous Albion Milk & Sulphur Toilet Soap.

For Maternity and Professional use. Price 11d. per tablet.

SAMPLES GLADLY SENT ON REQUEST.

THE ALBION SOAP Co. Ltd., 30/2 Thames St., Hampton, Middx.

The relief of
NIGHT CRAMPS
by
QUININE

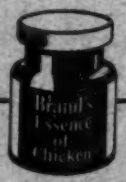
The painful condition of idiopathic night cramps of the extremities may seriously interfere with sleep. They can, however, be abolished in 9 cases out of 10 by the administration of small oral doses of quinine (3–5 grains) at bedtime.

Literature available on request

HOWARDS OF ILFORD

Makers of Quinine Salts since 1823

HOWARDS & SONS LTD · ILFORD · ESSEX



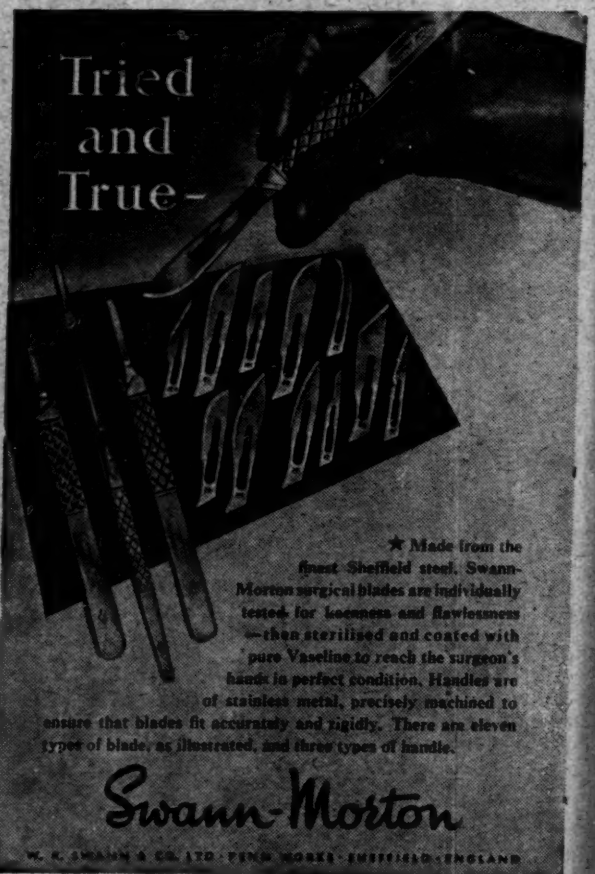
When
nourishment
is the main problem . . .

BRAND'S ESSENCE is a first-class protein of animal origin. Being partly hydrolized, it is capable of easy ingestion, digestion and absorption. It is extremely palatable, and may be taken either as a jelly or as a liquid. It helps to support convalescence and assists in restoring a positive nitrogen balance.

The major indications for the administration of Brand's Essence are loss of appetite during fatigue, acute infections, and dysphagia or digestive disturbances due to organic or bacterial lesions of the mouth, oesophagus and alimentary tract, and after surgical procedures.

The addition of Brand's Essence to low residue and weight-reducing diets is especially appreciated by the patient.

Brand's Essence
(BEEF OR CHICKEN)



★ Made from the finest Sheffield steel, Swann-Morton surgical blades are individually tested for keenness and flawlessness—then sterilised and coated with pure Vaseline to reach the surgeon's hands in perfect condition. Handles are of stainless metal, precisely machined to ensure that blades fit accurately and rigidly. There are eleven types of blade, as illustrated, and three types of handle.

Swann-Morton

W. K. SWANN & CO. LTD · PENN WORKS · SHEFFIELD · ENGLAND

BRITISH MEDICAL JOURNAL

LONDON SATURDAY JANUARY 10 1953

THE EPIDEMIOLOGY OF SOME SKIN INFECTIONS*

BY

ROBERT CRUICKSHANK, M.D., F.R.C.P., D.P.H.

Professor of Bacteriology, London University, at St. Mary's Hospital Medical School, London, W.2

Malcolm Morris was appointed as the first dermatologist to St. Mary's Hospital in 1881. It was an important year for microbiology, for in 1881 Koch described his methods for obtaining pure cultures of bacteria, Pasteur demonstrated the protective action of anthrax vaccines, Alexander Ogston, in Aberdeen, isolated and described the pyogenic cocci, while Raymond Crocker, in London, first saw "chains of micrococci" in the vesicle fluid of impetigo contagiosa. Malcolm Morris was the last child of a family of fifteen. He came from Bristol to St. Mary's at the age of 17, his interest in medicine having been stirred by an experience in hospital with a broken leg after falling into the docks. His early postgraduate life was spent in general practice in Yorkshire and later in London, but after four rather dissatisfying years he decided to be a specialist and set up his plate in Montagu Square as a dermatologist. Later he moved to Harley Street, where he practised until he retired at the age of 69 in 1918. He died in 1924.

Like many of the older dermatologists, and some of the younger ones, Malcolm Morris was a good general physician, and he loved to play the medical detective in dealing with rather obscure skin diseases. He was among the first to recognize the skin sensitivities to plants like poison ivy and the primula. In this Memorial Lecture, therefore, I make no apology for taking as my subject "The Epidemiology of Some Skin Infections," for I am sure Sir Malcolm Morris would have approved the use of the modern detective devices in tracing the sources and modes of spread of skin infections and so leading to better methods for their prevention and control.

From a large and fascinating field the four diseases with which I shall deal—pemphigus of the newborn, impetigo contagiosa, ringworm of the scalp and skin, and plantar warts—represent infections with a variety of parasites which range from the infinitely small and host-dependent viruses to the much larger and semi-independent fungi. Here is the first advantage of studying the epidemiology of skin infections, for not only does the skin act as host to a wide range of parasites but we can easily watch and study the development of the host–parasite relationship and not simply conjecture what is happening in some hidden corner of kidneys or lungs. The factors concerned in the initiation of infection are still largely unknown, and I believe there is a fruitful field for the observer who is prepared to study carefully the early stages of skin lesions in communities where infections like impetigo, pemphigus, and ringworm are spreading.

*The Malcolm Morris Memorial Lecture given at St. Mary's Hospital, London, on December 13, 1951.

Pemphigus in the Newborn

The first skin infection I wish to discuss is pemphigus in the newborn, or bullous impetigo. An infection of the first month of life, this condition develops as a small red area in the centre of which a bleb containing clear fluid rapidly forms. Within two to three days this fluid becomes turbid, and the bleb bursts, leaving a small red area surrounded by a frill of shreds of dead skin. The blebs occur most often where two skin surfaces rub together—for example, the groins, axillae, the inner surfaces of the thighs—and also on exposed surfaces which are liable to irritation, but never on the palms of the hands or soles of the feet. The condition to-day is usually mild, but there may develop a generalized exfoliative dermatitis or a secondary staphylococcal or streptococcal septicaemia, or the child who is puny and premature may succumb to other secondary infections. Outbreaks were not uncommon during the late war years for a variety of reasons—for example, emergency maternity homes with inadequately trained staff, poor quality soap, or lack of olive oil, which was replacing soap for cleaning the skin.

When we try to trace the source of infection we are immediately up against the real difficulty that the staphylococcus which causes this infection is the most ubiquitous of germs. It is present in the noses of half the adult population and on the skin of about a quarter; it is found in dust, in the air, and on blankets and various other inanimate objects that may come in contact with the baby's skin. Thus, while we might look for the source of infection in a septic sore on the finger of an attendant or in a breast abscess in one of the nursing mothers, we are often completely nonplussed regarding its origin. Fortunately, in recent years two technical advances have helped to trace the source of the epidemic strain. First, we can readily recognize the potentially dangerous staphylococci by the fact that they produce an enzyme, coagulase, which causes clotting of plasma. Secondly, we have been able to subdivide a high proportion of the pathogenic staphylococci into many different types by utilizing the activities of the bacterial parasite, bacteriophage, which selectively destroys the staphylococcus. (The techniques for demonstrating coagulase activity and phage-typing were shown in extracts from the film, "Tracing the Spread of Infection.") Using this phage-typing technique or the serological method developed by Cowan (1938, 1939), various workers have shown that the cases in any outbreak of pemphigus are usually

due to one epidemic strain of staphylococcus, but that in different outbreaks the epidemic strain may belong to different staphylococcal types.

In searching for the source of infection, Allison and Hobbs (1947) showed that the epidemic strain does not come from the mother's nose, but that a high incidence of nose-carriers of the epidemic strain among the nursing staff may precede the outbreak among babies. When it is known that a large proportion of nose-carriers also harbour the same staphylococcus on the hands, it is easy to see how the organism may become transferred to the baby. Outbreaks are not confined to maternity institutions, for in domiciliary midwifery we have been able to trace cases of infection to one individual midwife who was a nose-carrier of the epidemic strain.

We must be careful, however, not to be too ready to incriminate the nurse, even if she may seem to be the most likely source of infection. Parker and Kennedy (1949), in an outbreak at Carmarthen, failed to find a significant number of nose-carriers of the infecting strain among the nurses, whereas this strain was commonly present in the noses of healthy contact babies, which suggested an infant-to-infant spread as the most likely mode of transmission. Their findings may explain the difficulty in controlling pemphigus by merely isolating the clinically infected cases, but even more drastic measures, such as ward closure, may fail to eradicate the infection. We must remember to deal with contaminated dust, blankets, napkins, and the like before reopening an infected maternity unit, and in this connexion there is much to be said for the small four- to six-bed unit in which mother and child are kept together. I should like to deprecate the indiscriminate use of penicillin cream for dealing with skin infections in the infant nursery. Its main effect is to lead to a sharp increase in penicillin-resistant strains, and we have had recent experience of severe penicillin-resistant staphylococcal infections among patients and staff in maternity departments.

Investigations in the Far East

While pemphigus in its typical bullous and infectious form is confined to infants in the first month or so of life in this country, it occurs as an adult infection in more tropical regions, and was a source of much trouble among our troops in the Far East during and after the late war. An M.R.C. dermatological research team went out to Singapore to investigate these prevalent skin infections, and I am indebted to Dr. J. R. May for permission to mention some of the findings embodied in his M.D. thesis. Among British troops in Singapore and Hong Kong bullous impetigo was found in 9%, whereas non-bullous impetigo was present in only 2% of the cases. Tropical bullous impetigo is characterized by superficial blebs of 0.3–0.8 cm. diameter with a turbid vesicle fluid and very little surrounding inflammation or local adenitis. These lesions are found most often in the axillae, and, in contrast to impetigo in temperate climates, the face is usually spared. May isolated coagulase-positive staphylococci from all of 101 *unruptured* blebs in 68 cases and haemolytic streptococci from only two atypical cases. Streptococci were, however, isolated more often from late ruptured lesions.

The main predisposing factor to bullous impetigo in the Tropics is obviously the moist, sodden condition of the skin in the axillae resulting from continual sweating and lack of ventilation in the hot, humid climate. It has, indeed, been shown that persistent moisture of the skin lowers its natural germ-killing powers, but, in addition to this locally lowered resistance of the host, May's bacteriological studies indicated that certain specific strains of the staphylococcus, different from those found in the nose or other skin lesions, were associated with bullous impetigo. There was, in addition, some evidence in favour of case-to-case spread. Thus the condition is similar in many ways to pemphigus in the newborn, and it seems that the occurrence of this distinctive skin lesion depends on a low resistance of the host skin associated with a staphylococcus endowed with epidemic propensities.

Impetigo Contagiosa

We turn now to the controversial subject of impetigo contagiosa, seen in its most typical form among children, but occurring also in adults. The controversy, which has gone on for half a century or more, concerns the primary aetiological agent, some believing it to be the haemolytic streptococcus and some the staphylococcus, while a specific impetigococcus has also been postulated (Hope Simpson, 1941). Good epidemiology depends, among other things, on careful clinical observation, and it is perhaps clinical confusion that has led to different views about the aetiology of impetigo. There is, I believe, good clinical and epidemiological evidence for the existence of two types of impetigo, due respectively to the haemolytic streptococcus and to the staphylococcus. The clinical differentiation was probably first made in Jadassohn's clinic in Breslau (see Epstein, 1940), where each new case of impetigo was carefully studied by more than one member of the staff, and each dermatologist stated, from his clinical observations, whether he regarded the case as streptococcal or staphylococcal impetigo. The clinical diagnosis was later compared with the bacteriological findings, and these trained observers were soon able to get reasonable agreement between clinical and laboratory conclusions.

Streptococcal Impetigo

Streptococcal impetigo is characterized by lesions mostly of the face and scalp, with thick stuck-on crusts of yellowish honey-like or dirty colour. There is some surrounding inflammatory erythema and the regional glands are often swollen. The original bleb of streptococcal impetigo is so short-lived that usually it is not observed. On the limbs, streptococcal impetigo may penetrate into the true skin and produce crusted ulcerations which are called ecthyma.

Staphylococcal impetigo is primarily bullous in character and the blisters may persist for some days. After the blisters have ruptured, the lesions become covered with thin flat varnish-like crusts. Healing occurs centrally at the same time as the periphery extends centrifugally, giving a circinate appearance. There is, however, little surrounding erythema and regional adenitis is rare.

When we consider the epidemiological evidence there are again good grounds for believing in two distinct entities. Streptococcal impetigo is the classical type of impetigo contagiosa found among schoolchildren, and various workers have established its streptococcal aetiology by examination of the blister fluid in the early stages of the lesion. This infection has a sex, age, and seasonal distribution remarkably similar to that of scarlet fever. About three-quarters of the cases in schools occur among children aged 5–9 years. Its incidence is minimal in the summer months, rises sharply to a peak in the late autumn; after a temporary dip around Christmas, it rises still further in the early spring and then falls steeply from mid-April. It thus simulates closely and antedates by three to four weeks the seasonal curve of scarlet fever, and it is interesting to note that the epidemic wave has already begun well before the reopening of schools in September. It is not to-day a highly contagious disease, family spread being noted by Newman (1935) in only one-sixth of his cases, while classroom spread was quite rare, although there was a tendency for aggregation of cases in particular areas of the town of Southampton.

The late Dr. C. J. Thomas, for many years school medical officer for the London County Council, believed there was an association between impetigo contagiosa and scarlet fever in schools, and suggested that an antecedent attack of impetigo might protect against scarlet fever. Although there is evidence that the haemolytic streptococcus isolated from impetiginous lesions usually belongs to Lancefield Group A, I know of no investigation utilizing Griffith's typing to show whether or not an outbreak in a school is due to one epidemic strain. It may be noted here that patients with streptococcal skin lesions, particularly when

grafted on eczema, may shed very large numbers of haemolytic streptococci into the environment in a way similar to the heavy nose-carrier and may thus give rise to secondary cases of various types of streptococcal infection (Lemon *et al.*, 1948).

Impetigo contagiosa is a disappearing disease among schoolchildren. This statement is borne out by collected data supplied from the chief medical officer, Ministry of Education, and from figures kindly collected for me by the medical officer of health for London and by a number of medical officers of health throughout the country. Infection has in the past been associated more particularly with poor personal hygiene, although the disease also occurs in well-cared-for children. There is no doubt that a dirty skin is more friendly to the haemolytic streptococcus than a clean skin, and the marked reduction in incidence might therefore be attributed to the steady improvement in the health, nutrition, and cleanliness of our children. There has been a change in the severity of the infection. Impetigo contagiosa, as Tilbury Fox first described it in 1864, was a highly contagious disease characterized by fever, chills, and general malaise, and was sometimes followed by nephritis. Since then, like other streptococcal infections, there has been a steady decrease in the virulence of the infection, until to-day it is a mild as well as a disappearing disease.

Staphylococcal Impetigo

Staphylococcal impetigo was a prevalent skin infection among our troops during the war years and was the subject of several interesting studies during that period. Thus Bigger and Hodgson (1943) showed that the staphylococcus was present in pure culture in the early lesion, and that secondary streptococcal infection occurred increasingly with the age of the lesion. Sheehan and Fergusson (1943) confirmed the predominance of *Staphylococcus aureus* as the primary infecting agent, and were able to reproduce impetiginous lesions of varying degree with cultures of the isolated staphylococci and with blister fluid, but not with haemolytic streptococci recovered from the lesions. This infection, in contrast to streptococcal impetigo, has its greatest incidence in the summer months, and in civilian practice is more common in adults than in children. It occurs commonly in areas with hot dry summers, as in Central Europe and the Middle West of America. Irritation of the skin of the face is probably an important predisposing factor, for, besides its age and geographical distribution, its prevalence among unseasoned male Service personnel in the summer months indicates that unusual exposure to the sun plus shaving helped to initiate the infection. There is very little bacteriological or epidemiological evidence about its contagiousness. In a group of men whom I examined there was no history of case-to-case spread (Cruickshank, 1941), and I would suspect that most of the infections are autogenous from staphylococci harboured in the nose. There is need for detailed bacteriological study, including phage-typing, to determine the source of the infecting staphylococcus.

That a low tissue resistance may be a factor in impetigo is suggested by the relatively high incidence of seborrhoeic impetigo and dermatitis seen among the troops in the war years and also in civilian practice. In the group examined by Twiston Davies *et al.* (1945) 148 out of 200 cases of impetigo among troops were included in the seborrhoeic group. There is serological evidence that the closely related condition of sycosis barbae is an autogenous infection caused by the staphylococci carried in the patient's own nose, and that prevention of relapse in this infection may depend on elimination of the staphylococci from the nose (Hobbs *et al.*, 1947).

Summing up the evidence about impetigo, I suggest that there are two distinct entities—one streptococcal and the other staphylococcal—which can be differentiated on both clinical and epidemiological data. Streptococcal impetigo is a contagious infection of schoolchildren, with epidemiological features in common with scarlet fever and other streptococcal infections. Staphylococcal impetigo is dependent on an antecedent and predisposing skin irritation which lowers resistance, and the infecting organism is probably autogenous rather than exogenous in origin.

Tinea Capitis

Although ringworm of the scalp has been gradually brought under some measure of control by improved methods of diagnosis and treatment, particularly the use of Wood's lamp and *x*-ray epilation, it is still an unpredictable and to some extent uncontrolled infection. During the late war there was a widespread recrudescence of the disease, associated, perhaps, with the mixing of town and country children and the general lowering of sanitary standards. But even in the more normal post-war years quite large epidemics have been occurring in different localities, and some attempts have been made to learn more about the epidemiology of this troublesome infection. Here, again, accurate diagnosis is essential; yet, despite the stimulus of Sabouraud's great work *Les Teignes*, published in 1910, little attention has been given in this country to the cultivation and precise identification of the species of fungi that cause ringworm. It was fortunate in some ways that Professor Topley had earmarked some of the highly specialized microbiologists for the Emergency Public Health Laboratory Service, for at the outbreak of the war it took Dr. J. T. Duncan from his "ivory tower" at the London School of Hygiene into the West Country, where he came in close contact with fungous infections. In 1945 he published the first comprehensive mycological survey of ringworm and identified 490 cultures of dermatophytes, of which 200 were *Microsporum audouini* and 156 *Microsporum canis*.

The high incidence of ringworm infections derived directly or indirectly from cats and dogs was impressive, but a large proportion of the specimens came from the south coastal counties, where, in certain areas, *M. canis* infection was apparently endemic, to the virtual exclusion of the more common *M. audouini* infections. Duncan's work was continued by his daughter, Mrs. Jacqueline Walker (1950), who published her findings in a three-year survey (June, 1946, to September, 1949) of ringworm infections in Great Britain and Ireland; among 2,473 cultures of dermatophytes she had identified 15 species in the genera *Microsporum*, *Trichophyton*, and *Epidermophyton*, and has confirmed Duncan's earlier recognition of two types of *M. audouini* and three types of *M. canis*, the two fungi which together were responsible for more than three-quarters of all her cases. This subdivision of the common skin fungi should allow us to trace more accurately the sources and modes of spread of ringworm infection, and local authorities should be encouraged to make use of these new precision tools.

Epidemics of scalp ringworm are practically always caused by *M. audouini*, which is a strictly human parasite. The fungus mostly attacks schoolchildren between the ages of 5 and 10 years and is six to eight times more common among boys than among girls, possibly because the girls' longer hair protects the scalp against infection. Transfer of the fungus probably occurs in a variety of ways, since the infected brittle hairs break off and contaminate clothing, caps, seats, dust, etc. Moreover, the fungus may remain viable in the environment for periods of a year or more. The high incidence of primary infection in the back of the head has led to suspicion of the barber's clippers, but it may be due in part to the easier access of the fungus to the scalp in that area, where the hair is usually cut short. Symptomless carriers, detected only by the use of Wood's lamp, are sometimes found, and search should always be made for carriers when the infection is spreading in a school. There is no evidence that the louse is a carrier of the fungus. Little is known about the development of immunity following infection.

M. canis is a natural parasite of cats and dogs, and is most likely to produce infection, often clinically inapparent,

in kittens and puppies. Infection, once transferred to the child, can pass from child to child, but becomes attenuated in the process and dies out after four to six transfers. None the less, in endemic areas 70–80% of the cases are probably due to case-to-case spread. Although large epidemics like those due to *M. audouini* do not ordinarily occur, infection with *M. canis* is widely distributed throughout Great Britain in both town and country—for example, endemic foci occur in Coventry, Leeds, Leicester, Reading, and Portsmouth, while about half the cases of scalp ringworm in the London area are due to *M. canis*. In the epidemiological study of ringworm, recognition of the three types of *M. canis* is important, for the local distribution of these types remains undisturbed over a period of years, which suggests that the predominance of one type prevents the establishment of a new invader. Control of this type of ringworm obviously depends on the recognition and eradication of the animal reservoirs.

Other animal hosts of fungous infections are cattle (*T. discoides*) and horses (*T. equinum*); these cause infections of the skin rather than of the hair. Curiously enough, while the human cases derived from cattle ringworm occur mostly in farm workers, those from the horse have mostly been of urban origin. Mention should also be made of favus, which still occurs sporadically throughout the country. It is a disease of greater severity but much lower infectivity than ordinary ringworm, and is therefore a family rather than a community infection.

A most interesting feature of scalp ringworm is that it virtually disappears at puberty, and the most persistent infections will clear up spontaneously at the onset of puberty. The most rational explanation of this phenomenon is that given by Rothman *et al.* (1947), who showed that adult hair fat secreted by the sebaceous glands is much more fungicidal than the corresponding child sebum. It would seem, however, from later work by Kligman and Ginsberg (1950), that the differences in fungicidal action may be more quantitative than qualitative, since it is well known that there is a great increase in the secretion of sebum after puberty. This fungicidal activity is associated with certain saturated fatty acids contained in the hair fat, and this new knowledge has been utilized in the preparation of fungicidal preparations like undecylenic acid.

In attempts to control the spread of ringworm, a common procedure has been to exclude the affected children from school; this may lead to much educational loss to a child, in some cases up to 20% of its school life. In a recent study in Maryland, U.S.A., exclusion from school was not required provided the affected children wore close-fitting caps (Schwartz *et al.*, 1946). A systematic search for possible modes of transmission was made, and, while cinema seats were exonerated, the barber's tools, particularly the clippers, came under strong suspicion. Infected hairs were shown to persist in the clippers, and in most establishments there was no satisfactory attempt at sterilization of clippers, combs, and brushes. For this purpose I would suggest the use of formalin vapour liberated in an airtight chamber in the barber's shop, for it has remarkable properties in surface sterilization against all kinds of vegetative and sporing mico-organisms, fungi, and viruses.

Tinea Pedis

I want to deal briefly with tinea pedis, or athlete's foot. This is a fungous infection of varying prevalence among older schoolchildren and young adults, particularly those living a communal life—for example, school boarders, students, and troops. The main causative fungi are *Trichophyton interdigitale, T. rubrum,* and *Epidermophyton floccosum*. The infection usually begins in the plantar fold of the fifth metatarso-phalangeal joint, involves the interspaces and the plantar surfaces of the toes and the soles, and may extend to the sides of the feet, not quite to the ankles. This limited localization corresponds with the part of the foot where the skin is devoid of hair follicles and sebaceous glands. There is little tendency to secondary infection elsewhere in the body, and the condition usually responds to the modern fatty-acid preparations. The localized distribution of athlete's foot in adult life is therefore not contradictory to the view that the ringworm fungi are particularly susceptible to the fatty acids associated with the increased production of hair fat after puberty. It has been noted that among students the incidence of infection increases to some extent with age, and it has been suggested that sensitization from previous attack is more likely to be manifested clinically.

The epidemiological problem that creates controversy is whether the infection is exogenous or endogenous in origin; in other words, is it a contagious disease, or does the fungus live a saprophytic life on the skin and cause infection only when there is a lowered tissue resistance, rather like the pneumococcus in bronchopneumonia? In favour of the exogenous contagious view are the prevalence of the infection among those living a communal life in boarding schools, Army barracks, and student hostels; the isolation of different species of fungus at short intervals in the same individual (although mixed infections are not uncommon); and the predominance of one or other of the three common types in some surveys. On the other hand, the occurrence in different surveys of 1–3% of symptomless carriers, the frequency of the same type of fungus in recurrent attacks, and the remarkably constant ratio in which the three common types are found in groups of men at different periods suggest that the fungus may be present as a saprophyte until maceration of the skin from extensive sweating, warm humid climates, or frequent use of swimming-pools allows the fungus to produce clinical infection (Hopkins *et al.*, 1947). It is most important that this problem be resolved, for rational preventive measures will depend on satisfactory evidence in favour of one or the other view. The importance in the spread of this infection of bath-mats, showers, sandals, etc., in swimming-baths is still unproved, and the use of disinfectant foot-baths is probably of little value. Most benefit is likely to come from avoidance of the conditions that encourage excessive foot sweating or maceration of the skin, and the use of hygienic measures that will keep the skin of the feet in a healthy state.

There can be little doubt that fungous infections of the skin and hair are still unduly prevalent, and present many unsolved problems. Yet medical mycology is scantily taught and rarely practised in most medical schools in this country, in contrast to America, where departments of medical mycology are the rule rather than the exception. Although the systemic mycoses are not serious problems here, we should encourage the training of microbiologists in modern mycological techniques. I can assure you that we practise what we preach, for Dr. J. R. May, after some training in the Mycology Department of the London School of Hygiene, has been co-operating with our dermatology department in the identification by microscopical examination and culture of material from a variety of suspected fungous infections of skin, hair, and nail. The harvest after 21 months, if modest, is encouraging in that from 160 specimens a positive result was obtained by microscopical examination in 64 instances, while there have been 45 positive cultures representing seven different species of pathogenic fungi. I should add that good results will be obtained only when both the clinician and the bacteriologist are enthusiastic mycologists.

Plantar Warts

The plantar wart, or verruca plantaris, has become a troublesome skin infection among schoolchildren, for it is a chronic disease which may cause considerable pain and disability to the patient and has defied various efforts at prevention or control. It is caused by a virus, and characteristically consists of a single wart in the metatarsal area, where the flattened wart is often surrounded by thickened skin so that it may simulate a corn. The sex-and-age distribution is interesting, for plantar warts are found four to five times more often in girls than in boys; about two-

thirds of the cases occur in the age group 11–15 and a quarter in children aged 7–11, while the infection is rare in infant departments. When more detailed inquiry is made into the age distribution of cases attending foot clinics, as Dr. A. W. Forrest has done at Leyton (personal communication), it appears that the incidence rises to a peak in the 12–13 age group and thereafter falls sharply. Although there is evidence that the wart is a self-limited infection, this maximum incidence around puberty suggests that there may be a hormonal factor concerned in its pathogenesis.

The predominance of plantar warts among girls and the increasing incidence with school age has found various explanations—the more frequent attendance at the swimming-bath, increasing use of barefoot physical training and dancing, and the use of communal plimsolls. While contaminated floors and shoes are likely vehicles for the spread of what is undoubtedly an infectious disease, they do not seem to me to explain satisfactorily the peculiar age and sex distribution. There is need here for an inquiry into possible predisposing physiological factors and for more careful observation of the habits of schoolchildren and their relationship to the occurrence of what is a readily recognizable infection. Unfortunately, the wart virus has not yet been grown artificially, so that its distribution outside the body cannot be determined. Sometimes, however, the mode of spread of an infection can be demonstrated by blocking one or other of the possible channels, and some concerted study in this direction would probably lead to more knowledge about the spread and to more rational measures for the control of plantar warts.

Conclusion

I will conclude by reminding you that infections of the skin are still a major cause of absenteeism from school and from work. In a recent survey among schoolchildren skin diseases were responsible for an absence rate of 23 per 100 children, which was greater than the absence rate from any one of the specific fevers (D. D. Reid, personal communication). I have remarked on only a few of the common skin infections—we might have discussed scabies or acne, boils, wound infections or erysipelas, or some of the ill-defined conditions whose aetiology is still obscure. I hope the dermatologists and mycologists and medical officers of health will forgive a microbiologist for wandering into their fields, but I think they will agree with me that there are still some fascinating problems to be tackled. I am convinced that problems of this kind can be tackled and solved satisfactorily only by co-operative efforts between the clinician, the administrator, and the laboratory worker.

REFERENCES

Allison, V. D., and Hobbs, B. C. (1947). *Monthly Bull. Minist. Hlth Lond.*, **6**, 109.
Bigger, J. W., and Hodgson, G. A. (1943). *Lancet*, **1**, 544.
Cowan, S. T. (1938). *J. Path. Bact.*, **46**, 31.
—— (1939). Ibid., **48**, 169.
Cruickshank, R. (1941). *Lancet*, **2**, 275.
Davies, J. H. Twiston, Dixon, K., and Stuart-Harris, C. H. (1945). *Quart. J. Med.*, n.s. **14**, 183.
Duncan, J. T. (1945). *British Medical Journal*, **2**, 715.
Epstein, S. (1940). *Arch. Derm. Syph., Chicago*, **42**, 840.
Hobbs, B. C., Carruthers, H. L., and Gough, J. (1947). *Lancet*, **2**, 572.
Hopkins, J. G., Hillegas, A. B., Ledin, R. B., Rebell, G. C., and Camp, E. (1947). *J. invest. Derm.*, **8**, 291.
Kligman, A. M., and Ginsberg, D. (1950). Ibid., **14**, 345.
Lemon, H. M., Loosli, C. G., and Hamburger, M., jun. (1948). *J. infect. Dis.*, **82**, 72.
Newman, J. L. (1935). *J. Hyg., Camb.*, **35**, 150.
Parker, M. T., and Kennedy, J. (1949). Ibid., **47**, 213.
Rothman, S., Smiljanic, A., Shapiro, A. L., and Weitkamp, A. W. (1947). *J. invest. Derm.*, **8**, 81.
Schwartz, L., Peck, S. M., Botvinick, I., Leibovitz, A. L., and Frasier, E. S. (1946). *Publ. Hlth Bull., Wash.*, **294**, 1.
Sheehan, H. L., and Fergusson, A. G. (1943). *Lancet*, **1**, 547.
Simpson, R. E. Hope (1941). Ibid., **1**, 683.
Walker, J. (1950). *Brit. J. Derm. Syph.*, **62**, 239.

TRIETHYLENE MELAMINE IN HUMAN MALIGNANT DISEASE

RESULTS WITH ORAL ADMINISTRATION OF ENTERIC-COATED TABLETS

BY

EDITH PATERSON, M.B., F.R.C.P.Ed.

P. B. KUNKLER, M.B., M.R.C.P.

From the Christie Hospital and Holt Radium Institute, Manchester

AND

A. L. WALPOLE, Ph.D.

From the Imperial Chemical Industries, Ltd., Research Laboratories, Hexagon House, Manchester

Since 1950 reports have been published on the use of triethylene melamine (T.E.M.) as an alternative to nitrogen mustard in the palliative treatment of Hodgkin's disease, of the chronic leukaemias, and of various other neoplasms of the reticulo-endothelial system. The drug has been known as trisethyleneimino-*s*-triazine or 9500 in this country and as triethylene melamine or T.E.M. in the U.S.A.

In our first experiments with this compound it was shown to be a potent inhibitor of the growth of the Walker tumour in rats when given in aqueous solution by the intraperitoneal or intravenous route. When given by mouth, about 50 times the parenteral dose was needed to produce a comparable effect (Rose *et al.*, 1950). These results aroused our interest in the potentialities of the compound as an agent for the treatment of human malignant disease, and, with the possibility of clinical trial in mind, its pharmacology and toxicology were studied in animals of various species (Hendry *et al.*, 1951). In the course of these studies it was shown to produce a consistent temporary fall in the peripheral leucocyte count of the dog when given intravenously in appropriate dosage. On the other hand, when it was given to dogs by mouth—in gelatin capsules containing the dry crystalline product—the effect was very variable. Because of this it was decided to adopt the intravenous route in our first clinical trials of the drug, the results of which have already been reported (Paterson and Boland, 1951). Oral dosing, however, is simple and convenient, and the possibility of employing this route with T.E.M. was kept in mind.

While our early trials were still in progress we learned that this mode of administration had, in fact, been used in clinical trials in America (Karnofsky *et al.*, 1951), where the activity of the compound had been independently discovered (Lewis and Crossley, 1950; Burchenal *et al.*, 1950). It is clear from these reports, whether experimental or clinical, that a greater variation occurs in the response to the drug when orally administered than when given intravenously. Since T.E.M. is unstable to acids it seemed likely that the erratic response to oral dosing noted in the dog was due to variable inactivation of the drug in the stomach, and that this might also account in part for the variable response in man. The possibility of obtaining a more uniform response from enteric-coated tablets was therefore explored by comparing the effect of plain and enteric-coated tablets of T.E.M. given by mouth upon the peripheral leucocyte

count in dogs. The tablets used for this and for the subsequent clinical trials were supplied by the pharmaceutical research and service laboratory of Imperial Chemical Industries Ltd. They were available in strengths of 1 and 5 mg.

Animal Experiments

Four male and two female animals were used in the main experiment. Food was withheld for one day and the compound given in a single oral dose the following morning. Water was freely available to the animals at all times; they were fed in the usual way on the afternoon of the day of dosing. One dog was given plain tablets only, two were given enteric-coated tablets only, while the remaining three received, on separate occasions, both plain and enteric-coated tablets. Dosing was on a body weight basis. A total leucocyte count was carried out at one-day or two-day intervals on blood drawn from an ear vein. After each dose the animals' white blood counts were allowed to return to normal before the next dose was given. The results of these experiments are shown in Tables I and II.

TABLE I.—*Effects of Oral T.E.M. in Dogs*

Dog	Dose Total (mg.)	mg./kg. (approx.)	Leucocyte Count per c.mm. Initial	Minimum Recorded and Remarks
Plain Tablets (and dry compound in gelatin capsules)				
379	100	10 } In	23,100	8,470 at 17 days
380	100	12 } gelatin capsule	27,830	Dead overnight after being dosed
439	35	5·0	24,200	9,020 on day 8
439	6	0·5	24,750	No significant change
440	35	5·0	14,025	3,960 on day 3. Dog died on day 4 with negligible count
442	40	4·0	16,610	5,995 followed by prolonged low count
442	20	2·0	19,500	11,440 on day 10
454	6	0·5	19,140	440 on day 10. Dog died on day 11 with negligible count
Enteric-coated Tablets				
443	15	2·5	18,590	ca. 55 on day 3. Dog died on same day
442	15	2·0	13,970	1,815 on day 6
442	5	0·5	14,190	3,410 ,, ,, 9
455	5	0·6	19,580	11,165 ,, ,, 4
454	5	0·6	18,535	2,200 ,, ,, 8
439	5	0·5	24,420	4,235 ,, ,, 7
439	5	0·5	29,700	2,057 ,, ,, 8

TABLE II.—*Comparison of Plain and Enteric-coated Tablets in Same Dogs*

Dog	Type of Tablet	Dose mg./kg.	Leucocyte Count per c.mm. Initial	Minimum Recorded and Remarks
454	Enteric	0·5	18,535	5,225 on day 4
	Plain	0·5	19,140	440 on day 10. Dog died on day 11
439	Plain	5·0	24,200	9,020 on day 8
	Enteric	0·5	24,420	4,235 ,, ,, 7
	Plain	0·5	24,750	No significant change
	Enteric	0·5	29,700	2,057 on day 8
442	Enteric	2·0	13,970	1,815 ,, ,, 6
	,,	0·5	14,190	3,410 ,, ,, 9
	Plain	2·0	19,500	11,440 ,, ,,10
	,,	4·0	16,610	5,995 on day 2, followed by prolonged low count

It will be seen from these tables that with plain tablets or gelatin capsules the effect produced by the drug bears little relation to the dose. For example, dog 379 survived a dose of 10 mg./kg. and dog 439 a dose of 5 mg./kg., whereas dogs 440 and 454 died with doses of 5 and 0.5 mg./kg. respectively. It is possible that dog 454, which had had a previous treatment with enteric-coated tablets, may have died of a cumulative effect in spite of apparent recovery of the white-cell count.

The response to the drug in enteric-coated tablets is much more consistent. A single dose of 2.5 mg./kg. in this form proved fatal, but a dose of about 0.5 mg./kg. regularly produced leucopenia without a fatal issue.

Clinical Trials

A total of 44 patients have been treated, and this report concerns all except one patient who was moribund on admission and died the day after his treatment. The cases were advanced, most of them being untreatable by radiation, or having ceased to respond to radiation, to urethane, to nitrogen mustard, and to intravenous T.E.M.

Preliminary haematological investigations were always done, and in addition the standard tests to determine hepatic and renal sufficiency. Acute renal failure has not been recognized after the use of T.E.M., and where renal damage has been discovered by post-mortem examination the findings have been consistent with the primary disease, and in one case were explicable as a late effect of localized *x*-irradiation.

No dietetic restrictions were imposed on the patients at the time of administration of the drug.

Hodgkin's Disease

Twenty-two cases were treated with oral T.E.M. In each case the diagnosis was verified histologically. The effects in those cases are briefly summarized in Table III. In most instances about 0.3 mg./kg. was administered as a single dose. Most of the patients so treated complained of slight and transient nausea; in five vomiting occurred and in two diarrhoea. Patients who were given the drug over a longer period had no gastro-intestinal symptoms.

In over two-thirds of the 22 cases a satisfactory remission of symptoms has been seen. The general condition of the patient improved in all but five instances.

The effect on fever was studied in seven patients who had an elevated temperature on admission. Of these, six became afebrile and in one the fever was uninfluenced (Case 20). One patient who was initially afebrile developed fever after treatment, coincident with the development of a pleural effusion.

Enlarged lymph nodes decreased in size in most cases; decrease in the size of the spleen was more varied and occurred chiefly where the spleen was not greatly enlarged.

Skin itching subsided in most of the patients with this symptom, but in some of them the relief lasted for only one or two weeks.

Pleural effusions have shown little diminution in our cases; in one the effusion almost disappeared (Case 20); in two others the effusion required aspiration on many occasions and afforded an opportunity to find whether the drug injected intrapleurally at the time of aspiration would be effective in countering the pleural extension of the disease. The small experience did not lead us to regard this as a worth-while measure.

It is almost impossible to gain an accurate assessment of pain. However, in all patients admitted with pain, whether spinal, pleuritic, or abdominal, relief was reported; this was most convincing in the five patients suffering from the pain of spinal deposits.

Cases 3, 8, and 10 showed a steady rise in haemoglobin of over 2 g. per 100 ml. In the majority the haemoglobin level remained about the same. Case 9 showed a significant fall in the haemoglobin level 52 days after treatment, but by the 67th day the original level had been regained.

The total improvement in the patient and his symptoms has been of rather limited duration. Remissions of eight to twenty weeks have been regarded as worth while. Subsequent treatments in the same patients have generally yielded less satisfactory results. On the whole the effects on Hodgkin's disease of T.E.M. given orally are similar to those obtained by intravenous injection and closely resemble those of the nitrogen mustards. The side-effects on the haemopoietic system are also similar. Leucopenia, which has been defined as a fall in the total white count below 4,000 per c.mm., occurred in 13 out of the 22 cases. In six of these patients the leucopenia was serious; thrombo-

TABLE III.—*Hodgkin's Disease Treated with Oral T.E.M.*

Case No.	Sex and Age	Total Dose mg./kg.	Overall Time of Administration	Effect on Disease: General Condition	Nodes	Spleen	Itching	Other Manifestations	Duration of Benefit (Weeks)	Remarks
1	M 56	0·3	1 day	Improved	Decreased	Decreased	Decreased	Fever and mediastinal nodes decreased	12	Leucopenia
2	M 25	0·3	1 ,,	,,	,,	—	—	Fever and abdominal pain decreased	7	
3	M 67	0·3	1 ,,	,,	,,	—	—	Dyspnoea decreased	22 still well	Severe leucopenia
4	F 25	0·3	1 ,,	,,	,,	Decreased	—	—	8 ,, ,,	Leucopenia
5	F 23	0·3	1 ,,	,,	,,	—	—	—	9 ,, ,,	,,
6	F 32	0·28	1 ,,	,,	,,	—	—	—	8 ,, ,,	,,
7	M 56	0·3	1 ,,	Remained good	,,	Decreased	—	—	23 ,, ,,	,,
8	F 30	0·3	1 ,,	Improved	,,	—	Decreased	Abdominal mass unchanged	22 ,, ,,	3 doses within 6 weeks
		0·2	1 ,,	,,	Unchanged	—	Unchanged	,, ,,		
		0·1	1 ,,	,,	,,	—	,,	,, ,,		
9	M 63	0·2	1 ,,	,,	,,	Decreased	Decreased	—	18 ,, ,,	
10	M 69	0·3	1 ,,	Remained good	Decreased	—	Unchanged	—	14 ,, ,,	Severe leucopenia
11	F 39	0·26	17 days	Improved	,,	—	—	—	12	
12	M 55	0·35	8 ,,	,,	,,	Unchanged	Decreased	Fever and spinal pain decreased. Bone lesions unchanged	6	Leucopenia
13	F 64	0·3	1 day	,,	Unchanged	—	—	Fever and abdominal mass decreased	Uncertain owing to subsequent *x*-ray therapy	,,
14	M 41	0·2	1 ,,	Improved	,,	—	Decreased	Spinal pain decreased	7	Interval of 7 weeks between doses
		0·2	1 ,,	Unchanged	,,	—	,,	,, ,,	1	Severe leucopenia followed
15	M 21	0·18	10 days	Improved	,,	—	—	Chest pain decreased	8	Leucopenia
		0·3	1 day	Unchanged	,,	—	—	—	Nil	Leucopenia; died 1 month thereafter
16	M 36	0·06	4 days	Improved	Decreased	Unchanged	—	Chest effusion unchanged	6	Severe leucopenia; died 5 months thereafter
17	M 16	0·25	3 ,,	,,	Improved	Unchanged	—	Fever decreased	5	2 doses within 1 month
		0·4	3 ,,	Remained good	,,	Improved	—	—	Uncertain owing to intercurrent disease	
		0·4	3 ,,							
		0·4	3 ,,	Remained poor	—	—	—	Fever decreased	Nil	Severe leucopenia
18	M 55	0·3	1 day	Remained good	—	—	—	Spinal pain decreased	6 still well	
19	F 24	0·3	1 ,,	Improved	Unchanged	—	—	Chest effusion unchanged	8	Died 4 months thereafter
20	F 17	0·8	34 days	Unchanged	Decreased	Unchanged	Decreased	Pleural effusion and spinal pain decreased. Bone lesions and fever unchanged	Nil	Severe leucopenia
21	M 24	0·42	14 ,,	,,	Unchanged	,,	—	Fever decreased	,,	Died 3 weeks thereafter
22	M 44	0·22	3 ,,	,,	—	—	—	Jaundice and oedema unchanged	,,	

TABLE IV.—*Chronic Lymphoid Leukaemia Treated with Oral T.E.M.*

Case No.	Sex and Age	Initial W.B.C.	Dose mg./kg. Initial Course	Lowest W.B.C.	Total Dose mg./kg.	Effect on Hb	Effect on Spleen Size	Effect on Node Size	Clinical Result
23	M 73	204,000	0·50	2,800	0·50	Rise	Decrease	Decrease	Satisfactory remission 8 months. Remains well. W.B.C. 28,000
24	F 55	223,000	0·18	21,400	0·48	,,	Unchanged	Unchanged	Remission 2 months. Subsequent irradiation, with remission 4 months
25	M 75	23,500	0·10	3,300	0·10	,,	Decrease	Increase	Satisfactory remission 5 months. Recent condition unknown
26	F 66	88,500	0·20	66,000	0·40	,,	,,	Decrease	Satisfactory remission 6 months. Remains well. W.B.C. 32,000
27	F 61	65,000	0·10	71,000	0·40	,,	Unchanged	,,	Satisfactory remission 4 months. Remains well. W.B.C. 10,000
28	F 80	18,000	0·10	6,700	0·10	,,	,,	—	Follow-up under 3 months
29	M 60	16,000	0·15	3,300	0·30	,,	Decrease	Unchanged	Remission 4 months. Deteriorating. W.B.C. 10,000

TABLE V.—*Chronic Myeloid Leukaemia Treated with Oral T.E.M.*

Case No.	Sex and Age	Initial W.B.C.	Dose mg./kg. Initial Course	Lowest W.B.C. After Initial Course	Total Dose mg./kg.	Effect on Spleen Size	Clinical Result
30	F 54	189,000	0·40	8,900	0·80	Decrease	Remission 6 months. Remains well. W.B.C. 11,000
31	F 58	89,000	0·60	14,900	0·90	,,	Remission 5 months. Remains well. W.B.C. 26,000
32	F 37	25,400	0·10	12,000	0·20	Increased	Died 4 months
33	F 44	54,000	0·15	7,000	0·55	,,	,, 3 ,,
34	F 58	47,000	0·40	36,000	0·40	Unchanged	,, 1 month
35	F 64	249,000	0·55	2,900	1·35	,,	,, 7 months
36	F 64	198,000	0·45	26,500	0·95	—	,, 7 ,,

cytopenia and petechial haemorrhages occurred, necessitating blood transfusions. The response to repeated transfusions of fresh whole blood has been good.

These six cases have been studied in the hope of illuminating the conditions under which severe leucopenia may occur. In one case no explanation could be advanced for the severe response. In another (Case 20) the occurrence was most likely due to the large dosage administered (0.8 mg./kg. in 34 days). Two patients had low polymorph counts before treatment; Case 3, for example, had a polymorph count of 2,500, although the total white count was 8,700. Case 16 also had an initial polymorph count of 2,100, with a shift to the left. Because his total white count was less than 4,000 the dose given was exceedingly small (0.06 mg./kg.); nevertheless, a severe leucopenia followed. Over a period of eight years this patient had had repeated *x*-ray treatments to enlarged lymph nodes and had been treated with nitrogen mustard one year before the treatment in question. His leucopenia was of long standing, but had never been severe with previous treatments.

In two cases the leucopenia might be ascribed to a cumulative effect. Case 14 was given two treatments with a seven-weeks interval. No leucopenia followed the first dose, the blood count being apparently satisfactory. The second dose of 0.2 mg./kg. produced the most serious leucopenia and thrombopenia we have encountered. This was shown in the bone marrow as a total reduction of cellularity. Case 17 developed leucopenia after the fourth dose within five months. Prior to this dose the polymorph count was high. The condition of the patient had, however, deteriorated. Further work is required to determine more accurately the factors responsible for these unexplained responses.

On the basis of these observations we now feel it is inadvisable to use T.E.M. in patients who have a low initial polymorph count.

From the point of view of avoiding severe leucopenia there has not seemed to be any real advantage in spreading the dose over several days, since the effects on the white-cell count do not become apparent until an interval has elapsed which has its median between the 14th and the 17th day after treatment. The cases which developed severe leucopenia did not show any shortening of this period, and the range of intervals was the same for those which had only a moderate effect on the white-cell count. This median interval of 14 to 17 days and the range found are strictly comparable to those following intravenous administration of the drug at appropriately lower doses.

In reviewing the cases it seems that a single dose of 0.3 mg./kg. should not be exceeded even if patients are under careful supervision. A slight reduction in the dose to 0.25 mg./kg. might be an advantage in allowing for the sensitive individual. At 0.3 mg./kg. more than half the patients will show leucopenia, in most cases slight. It is inadvisable to use the drug if the initial polymorph count is low.

For the management of our patients we have generally admitted them to hospital. The drug has also been given to out-patients; in these circumstances the patient has reported back at weekly intervals for a blood count and has been told that he should be prepared to come into hospital should the white-cell count be unsatisfactory.

Chronic Leukaemia

Seven patients with lymphoid leukaemia and seven patients with myeloid leukaemia have been treated by oral T.E.M. Summaries of the results in these cases are given in Tables IV and V. There seems to be a wide variation in the sensitivity of the leucopoietic system to T.E.M. in the chronic leukaemias, particularly in the lymphoid type.

In the first patients treated a daily dose of 1 mg. was given until a response in the blood picture was noted. This method was abandoned when it became apparent that a severe drop in the white-cell count and platelet count might follow even when the administration of the drug was stopped as soon as the white-cell count began to fall. Latterly it has been our practice to administer a dose of 0.1 to 0.2 mg./kg. and to follow this by similar doses, given at intervals determined by the response. The interval between treatments should not be less than 14 days.

During the initial treatment with T.E.M. patients have remained under observation in hospital, but for subsequent maintenance treatment they have attended at monthly intervals. It has been thought unwise to let patients take supplies of this drug home, and each dose has been given in the light of the patient's clinical and haematological condition.

Chronic Lymphoid Leukaemia

There was a definite symptomatic improvement in five out of the seven patients with lymphoid leukaemia; in four the spleen was reduced in size and in three the nodes became smaller.

No attempt has been made to keep the total white-cell count within normal limits. The decision to give or withhold treatment has depended as much on the symptoms as on the leucocyte count. With this mode of treatment erythropoiesis does not seem to have been adversely affected and, as will be seen in Table IV, the haemoglobin levels have increased.

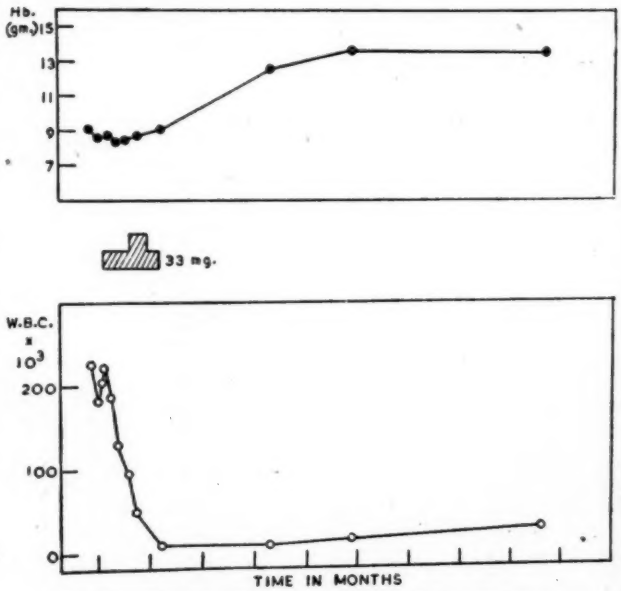


FIG. 1.—Case 23. Haemoglobin and white-cell counts.

Case 23 (Fig. 1).—A man aged 73 was first seen at this hospital with large nodes in all areas; these had been present for eight months. The spleen was enlarged to the umbilicus. Haemoglobin, 8.6 g./100 ml.; W.B.C., 350,000 per c.mm., mainly lymphocytes. He received single exposures of *x* rays to each side of the neck and each axilla, which resulted in a slight reduction in the size of the nodes in these areas. Four months later he developed signs of superior mediastinal obstruction. He was treated with an initial course of T.E.M. to a dose of 0.5 mg./kg., and this resulted in a fall in the W.B.C. from 204,000 to 2,800 per c.mm. in 58 days. During this period the largest neck node was reduced from 5 to 2 cm. in diameter, axillary nodes 5 and 6 cm. in diameter became impalpable, and the spleen was reduced in size. The dyspnoea and dizziness initially present disappeared and the mediastinal nodes were shown on radiological examination to have decreased in size. Eight months after treatment he was without symptoms but the nodes were again increasing in size. Haemoglobin, 13.6 g./100 ml.; W.B.C., 28,000 per c.mm.

It is probably fair to conclude that the result obtained with T.E.M. in this case was superior to that which would have been obtained by further radiotherapy, since the local response in the nodes to single exposures of 400 r and 800 r was disappointing.

T.E.M. is not, however, always superior to *x*-irradiation, as Case 24 illustrates. The dose of T.E.M. was probably low, but in our opinion this does not explain the longer remission obtained by splenic irradiation at a later stage of the disease.

Chronic Myeloid Leukaemia

Five out of the seven patients treated have died. They were all advanced cases that had already been treated with *x* rays or urethane, or both. At the best only transient remissions were obtained with T.E.M. In two patients who had not been previously treated, maintenance treatment has resulted in clinical and haematological improvement.

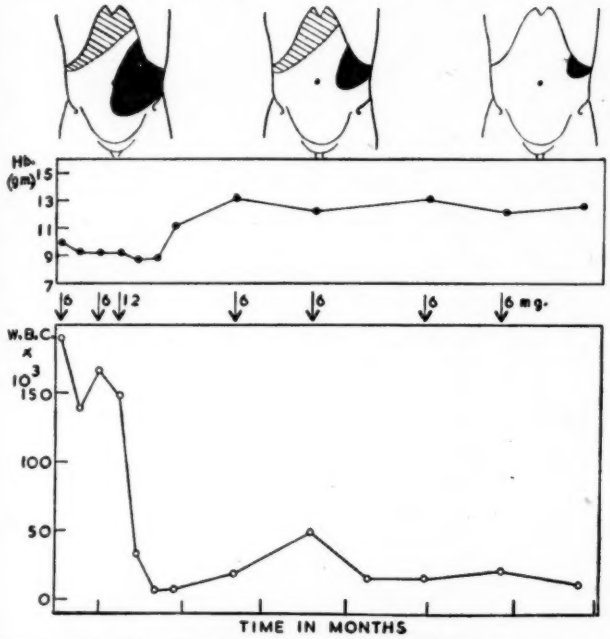


FIG. 2.—Case 30. Showing reduction of size of liver and spleen, and haemoglobin and white-cell counts.

Case 30 (Fig 2).—A woman aged 54 presented with lassitude, cramp in her legs, and a painful lump in her left loin. On examination the main findings were pallor, a palpable liver, and a spleen extending 7 cm. below the umbilicus. Haemoglobin, 10 g./100 ml.; W.B.C., 189,000 per c.mm.—polymorphs 127,500, lymphocytes 10,500, mononuclears 2,000, eosinophils 8,000, basophils 8,500, blasts 5,500, promyelocytes 13,000, myelocytes 9,500, metamyelocytes 4,500. The initial course of T.E.M. consisted of a dose of 0.4 mg./kg. given over 21 days. On discharge from hospital six weeks after admission the white-cell count was 8,900 per c.mm. and the haemoglobin 11.2 g./100 ml. The spleen extended only a centimetre below the umbilicus. She was free from pain. Maintenance treatment with T.E.M. has been given, and when last seen six months after the beginning of treatment the total white count was 11,100 per c.mm. and the haemoglobin 12.7 g./100 ml. The spleen had been reduced further in size, and was now confined to the left upper quadrant of the abdomen. The liver was not palpable.

Case 31.—A woman aged 58 was first seen at this hospital complaining of swelling of the abdomen for about a year and lassitude for three months. The spleen was extended below the umbilicus and the liver was palpable. Haemoglobin, 9.3 g./100 ml.; W.B.C., 89,000 per c.mm. The differential count and the sternal marrow confirmed the diagnosis of myeloid leukaemia. T.E.M. was given in repeated doses of 0.1 mg./kg. after an initial course of 0.6 mg./kg. given over 51 days. Since discharge from hospital maintenance therapy has been pursued. When last seen five months after beginning treatment she was symptomatically improved and the spleen was much reduced in size. Haemoglobin, 11.8 g./100 ml.; W.B.C., 26,000 per c.mm.

It is clear that the results obtainable in advanced cases of myeloid leukaemia are poor. The two satisfactory cases (Nos. 30 and 31) were previously untreated; it is just in such patients as these that splenic irradiation or urethane treatment leads to remissions of many months.

Miscellaneous Types of Malignant Disease

No beneficial effect was obtained from the use of oral T.E.M. in two cases with carcinoma of the breast, in both of which secondary deposits continued to grow after treatment. One of these cases was given a very high total dosage of 0.9 mg./kg. spread over 22 days. No significant leucopenia occurred; the haemoglobin was unchanged and the platelets remained abundant.

Two patients with proved radio-resistant tumours also failed to respond. One had widely disseminated malignant synovioma; the other was a moribund patient with metastases from a malignant teratoma of the testis.

The only patient with polycythaemia vera was given a single dose, 0.25 mg./kg., and did not show any reduction in the red-cell count over the following six weeks. He was then given P^{32} and responded after two doses each of 5 mC, the reduction occurring eight weeks after the initial dose. Lack of response to T.E.M. in this case is contrary to the results obtained in other cases with intravenous use of the drug. It is possible that the dose was insufficient or that enough time was not allowed in which to observe the effects.

The only cases apart from Hodgkin's disease and chronic leukaemia in which we have observed some benefit were both reticulo-endothelial in type. One was a case of a typical lymphofollicular reticulosis in which a dosage of 0.25 mg./kg. given over three days produced a regression of lymph nodes in the mediastinum and elsewhere, and atelectasis of a lung lobe slowly cleared up. Another patient with a generalized reticulosis, which could not be classified, showed a partial response in the mediastinal nodes after a single dose of 0.3 mg./kg.

Summary and Conclusions

This contribution to the palliative treatment of chronic malignant disease is an extension of the investigation of triethylene melamine, which has been used both intravenously and orally with moderate success in certain diseases.

The clinical trial of a new substance presents difficulties. One is the very advanced stage of the disease in those patients selected for treatment. Another is the choice of dose level; this is not infrequently discovered in retrospect to have been inadequate or injudicious. Nevertheless the trial of enteric-coated tablets has revealed a reasonable consistency between dose and effect when dosage is given on a weight basis.

A beneficial effect has been obtained in Hodgkin's disease, in chronic leukaemia, and in two other cases of reticulo-endothelial disease; no good effect has been obtained in the tumours of epithelial or connective-tissue origin. This finding corroborates previous reports on the scope of the drug when it was administered intravenously at suitably lower dosage.

In Hodgkin's disease we have found that a dose between 0.2 and 0.3 mg./kg. given either undivided or spread over two or three days is suitable. Treatment should not be given in the face of a leucopenia induced by previous treatments by other agents. Repetition of the treatment is possible, provided that the polymorphonuclear leucocyte count is fully restored.

The possibility of latent damage to the haemopoietic system should be borne in mind; this was illustrated by the fate of one of the experimental dogs given a second treatment and by the serious leucopenia which occurred in one of our cases of Hodgkin's disease after a second small dose.

In chronic leukaemia the dosage has been administered at a lower level of 0.1–0.2 mg./kg. and has been repeated at intervals of several weeks for maintenance.

It has been notable that the response of cases of lymphoid leukaemia has been better than that of the myeloid cases. This is probably true even when allowances are made for the small numbers and the varied clinical condition of the patients. This finding has been encouraging, since with most other therapeutic agents the response of lymphoid leukaemia has been less favourable than that of myeloid leukaemia.

Tests of renal and hepatic function have not revealed changes after treatment, and post-mortem examinations, although often demonstrating long-standing damage or infiltration by disease, have not shown signs which could be definitely interpreted as due to toxic effects from treatment.

The haemopoietic system is highly sensitive to triethylene melamine, the effects being seen on the lymphocytes, granulocytes, and platelets of the peripheral blood.

The simplification of dosing by oral administration of enteric-coated tablets has seemed to be a worth-while advance; nevertheless by its very simplicity it carries a certain danger of being used without the essential haematological controls.

REFERENCES

Burchenal, J. H., Crossley, M. L., Stock, C. C., and Rhoads, C. P. (1950). *Arch. Biochem.*, **26**, 321.
Hendry, J. A., Homer, R. F., Rose, F. L., and Walpole, A. L. (1951). *Brit. J. Pharmacol.*, **6**, 357.
Karnofsky, D. A., Burchenal, J. H., Armistead, G. C., Southam, C. M., Bernstein, J. L., Craver, L. F., and Rhoads, C. P. (1951). *Arch. Intern. Med.*, **87**, 477.
Lewis, M. R., and Crossley, M. L. (1950). *Arch. Biochem.*, **26**, 319.
Paterson, E., and Boland, J. (1951). *Brit. J. Cancer*, **5**, 28.
Rose, F. L., Hendry, J. A., and Walpole, A. L. (1950). *Nature, Lond.*, **165**, 993

TUBERCULOSIS CASE-FINDING BY THE TUBERCULIN-TESTING OF INFANT-SCHOOL CHILDREN

BY

I. A. MacDOUGALL, M.R.C.S., L.R.C.P., D.P.H.
Medical Officer of Health and School Medical Officer, Bournemouth

J. R. MIKHAIL, M.R.C.S., L.R.C.P.
Assistant Chest Physician, Bournemouth

AND

W. H. TATTERSALL, M.D.
Consultant Chest Physician, Bournemouth

"On numerous occasions, only the tuberculin reaction of a child or adult has revealed unsuspected infectious cases among his associates."—J. A. MYERS, Minnesota.

Several years ago one of us (Tattersall, 1946) expressed the opinion that tuberculin-testing of young children should be more extensively used for case-finding in this country. Several interesting cases of tuberculosis had been discovered in this way. For example, the mother of two tuberculin-positive children was able to give a thorough account of the social contact of her daughters aged 5 and 3, which suggested that the only association they had had with anyone liable to cough was their father, who for the past year had been in Hong Kong. A message was sent advising that he should have his chest *x*-rayed, and he was invalided home with consumption.

This experience, after various trials and errors, led to the more extensive tuberculin survey of all the infant-school entrants in Bournemouth during the Michaelmas term of 1951, on which a preliminary report has been published (MacDougall, Mikhail, and Tattersall, 1951). Circumstances for this work had become propitious: the mortality rate from all forms of tuberculosis in Bournemouth during 1950 had fallen to 33 per 100,000 of population, the milk cleanliness in the town was known to have been good for a number of years, and the whole-hearted co-operation of all parties concerned could be anticipated. It was decided to test about 2,000 children over a period of about ten weeks which would cover almost all the children in the infant schools who had not yet reached their sixth birthday, including all the school entrants.

Since 1947 at least 97% of the milk sold in this county borough has been heat-treated, pasteurized, or from tuberculin-tested herds, and by the end of 1951 100% pasteurization had been attained. During the past five years all milk samples tested have complied with the current prescribed standards under the regulations of the Milk Special Designations Act, 1949; and under the Food and Drugs (Milk, Dairies, and Artificial Cream) Act, 1950, the town is shortly to become a "specified area" for the retail of "designated" milk. It therefore seemed reasonable to assume that a child beginning school and found to be tuberculin-positive would have been infected from a human source. The majority of such children would have led relatively sheltered lives, being too young to have had many casual stray and unknown contacts, so that any child thus infected would probably have incurred the infection within its own immediate family circle. The Michaelmas term survey was therefore designed to seek out and examine as exhaustively as possible all the relatives and friends (especially the adults) of tuberculin-positive school entrants.

A second survey during the Lent term was designed for comparison with the Michaelmas survey. A comparable number of infant-school children were tested, and their contacts examined, but by a slightly different procedure. This paper describes the methods and results of both surveys.

The Tuberculin Test

It was thought that the Mantoux test would require the positive written consent of a parent; so it was decided to proceed with the jelly test and advise parents that the test would be applied unless they objected. This was amply justified, since the success of the work depended on the co-operation of the family contacts of tuberculin-positive children. The technique used was that described by Dick (1950), and adopted for the Medical Research Council National Tuberculin Survey (1952), although later, suspecting the incidence of positive reactors might be unduly low, a slight modification recommended by Lendrum (personal communication) was adopted—namely, reading the results on the fourth day instead of the third day after application of the test. Flourpaper abrasion of the skin was not used on children under 6 years of age. In the Michaelmas survey whenever the result of the jelly test was doubtful a Mantoux test was performed, so that we always had a definite result; parental consent for this was obtained at the reading of the jelly test.

Organization of the Michaelmas Survey

1. The medical officer of health obtained the permission of the Education and the Health Committees for the investigation, and the scheme was explained to the head teachers of the schools involved.

2. The Public Health Department forwarded printed letters in stamped sealed envelopes to the school, from which

they were addressed to the parents of the appropriate children and posted a week before the test was to be applied.

3. The following form of letter was evolved :

Dear Sir/Madam,

It is proposed to undertake the tuberculin-testing of the school children in the Borough who are attending infants' departments. The test will take place at .....on.....

The action taken consists in placing a little special jelly on the child's back and strapping it in position for two days. It does not involve any injections and should not upset your child in any way.

Please remove the plaster on.....and attend at the school on.....for your child's back to be examined. Please do not allow the child to have a bath while the plaster is in position.

Unless I hear from you to the contrary, I will assume that you agree to this procedure. (Your child may already have been tested, but I hope you will be agreeable to a further test being carried out.)

You are invited to be present on the..... morning, when the test is applied, and on the..... morning, when the result is read.

The result will be communicated to you, along with any observations, in due course.

If you desire any further information I suggest that you get in touch with me.

Yours faithfully,
I. A. MACDOUGALL,
School Medical Officer.

4. At the same time the Public Health Department sent to the schools special tuberculin-test record cards which fit into the standard school health record card. Names and addresses were filled in on these cards by the school staff at the time of sending the initial letter to the parents.

5. Any objection to testing led to the removal of that child's tuberculin record card.

6. The schools were visited on Monday mornings by one of us (J. R. M.), accompanied by the school nurse for that school. It was found convenient to tuberculin-test about 200 children at a session ; this produced suitable numbers at each stage of the investigation. A ten-minute explanatory talk was given to the assembled parents, usually the mothers —generally about 80% of the children were accompanied— pointing out that tuberculosis is spread by germs, explaining the significance of pasteurization of milk, and emphasizing the importance of finding any infectious cases. It was stressed that the duty of bringing all the contacts of a child with a positive reaction fell on the parents, and that only by their co-operation could the health of their child be protected. We believe that this brief address, by winning the co-operation of the family from the start, is the most important detail in successful case-finding.

7. Children were tested in alphabetical order by classes, the help of the class-mistress being invaluable in keeping the children in this order while the parents helped with the undressing and dressing of the children. Specially prepared small plasters—2 by 1½ in. (5 by 3.8 cm.)—with gauze backings were made for covering the jelly, and this additional facility in application amply justified the slight extra cost. Two hundred tests could be applied without undue haste in one and a half hours.

8. The school was revisited on the following Friday and the tests were read. In all cases where the parents were not present a printed letter giving the result was posted to them. It was found in practice, however, that nearly all the positive reactors were accompanied on the day the test was read. A further explanatory talk was then given, stressing the importance of the child being immediately *x*-rayed and the parents' responsibility for finding who might have infected their child. The importance of talking to the parents cannot be overemphasized ; it was found that with their co-operation all the tuberculin-positive children and their family contacts, often neighbours as well, readily attended for fluoroscopy ; thus the number of follow-up visits to be made by the health visitors became trifling.

9. Once the mothers' anxiety has been aroused it should be alleviated as soon as possible. Therefore, when a positive reactor was found, fluoroscopy appointments were given to the mother for each member of the household and any other friends or frequent visitors that the mother could name as seeming relevant. The health visitor listed all these persons, which information was kept on the child's tuberculin record card. If the mother was able to account for the child's infection by contact with a known case, the alleged patient's name and address were taken for confirmation. Usually this proved correct.

10. After the tuberculin tests had been read at the school the record cards were returned to the Public Health Department.

11. Although several fluoroscopy sessions were available during the following week, it was found that 90% of the contacts attended the session on the morning after the reading of the test. All the children and their contacts were screened, following the technique described by Hall and Tattersall (1950), and the results of that examination were told to the parents immediately. Only those exhibiting any fluoroscopic abnormality were filmed. The completed fluoroscopy reports were forwarded to the Public Health Department and entered on the tuberculin-test record cards. Letters were sent by the department informing each general practitioner concerned of the *x*-ray result.

12. All other children attending the clinic as contacts were tuberculin-tested, and this measure led to the discovery of two cases which developed tuberculous meningitis less than three months later.

Organization of the Lent Term Survey

The results of the Michaelmas investigation were considered successful enough to justify corroboration by a further survey. It was decided to tuberculin-test the remainder of the infant-school population, a comparable number of children, though on average slightly older. The whole of this second survey was carried out in a fortnight during the Lent term, and differed from the Michaelmas survey in two essential ways. First, all the tuberculin-testing was performed and read entirely by the school nurses, working in pairs. Having assisted in the Michaelmas survey, they were familiar with the procedure. Secondly, by courtesy of Dr. J. S. Robertson, the medical director, the tuberculin-positive children and their contacts were examined by the mass radiography unit.

The application and reading of the tuberculin tests were the same, but contacts were given appointments to attend the mass radiography unit and passed through their routine in the usual way. However, special sessions were arranged so that men, women, and children could attend as a family group, and they were photographed clothed. This arrangement did slightly slow down the ordinary rate at which examinees passed through. The *x*-ray appointment form was clipped to the mass radiography card, and when the miniatures were read the *x*-ray forms were completed by the medical director and returned to the Public Health Department. All abnormal films were dealt with by the usual mass radiography routine. A nurse attended these sessions to tuberculin-test any other children attending as contacts.

Results

The numbers of children eligible, tested, and tuberculin-positive are shown for both surveys in Table I.

TABLE I.—*Children Tuberculin-tested*

	Michaelmas Term	Lent Term
Eligible children	2,123	2,161
Refused	70 (3%)	79 (3·7%)
Absent	188 (8%)	149 (7%)
Number tested	1,865	1,933
Positive	42	135
Doubtful result	0	28

The number and percentage of positive results of the tuberculin tests are shown by age groups for the Michaelmas and Lent terms in Table II.

TABLE II.—*Number and Percentage of Positive Results of Tuberculin Tests in Age Groups*

Age Groups	Michaelmas Term		Lent Term	
	Total No. Tested	Total Positive	Total No. Tested	Total Positive
3 years	99	0	18	1
4– ,, ...	589	14 (2·4%)	135	6 (4·4%)
5– ,, ...	1,177	28 (2·4%)	271	16 (5·8%)
6– ,, ...	—	—	558	46 (8·2%)
7– ,, ...	—	—	753	68 (9·0%)
8– ,, ...	—	—	182	23 (12·6%)
9– ,, ...	—	—	16	3

It will be noticed from these data that the incidence of positive reactors in each age group is rather low when compared with the findings of the National Tuberculin Survey in the urban areas of Southern England. However, on closer scrutiny of the available comparable data the findings are acceptable. Bournemouth is a well-housed seaside resort with very little industry, and in many ways more comparable to a rural area than an urban one. Table III gives

TABLE III.—*Percentage of Tuberculin-positive Reactors in Young School Children Found in Recent Surveys*

Place	Survey*	Method	Age Groups				
			4 yrs.–	5 yrs.–	6 yrs.–	7 yrs.–	8 yrs.–
Bournemouth ..	(1)	Jelly	2·7	3·0	8·2	9·0	12·6
Radnor	(2)	,,	—	3·5	2·9	5·0	5·4
Portsmouth ..	(3)	,,	—	5·1	8·7	10·7	15·2
Manchester ..	(4)	Mantoux 10 T.U.	—	4·3	14·1	20·6	16·3
Montgomery ..	(3)	Jelly	—	8·4	8·7	12·4	15·9

* (1) The present investigation.
(2) Jones Davies (1950), using Jensen's jelly preparation.
(3) Medical Research Council National Tuberculin Survey, 1949–50 (1952).
(4) Wolman (1952) using 1/1,000 Mantoux test on primary-school children in a working-class area of Manchester.

the incidence of tuberculin-positive reactors in primary-school children as reported in several recent investigations.

The origin of the tuberculous infection of the tuberculin-positive children is indicated in Table IV. This table also shows the number of contacts examined in each group of both the Michaelmas and the Lent term surveys, and suggests, what perhaps seems obvious, that the greater the number of contacts examined the greater the likelihood of case-finding success. Nevertheless, in 14 of the 17 new cases of adult disease the patient was a parent of the tuberculin-positive child.

TABLE IV.—*Sources of Infection of the Positive Reactors and the Numbers of Contacts Examined in Each Group of Children*

	Michaelmas Survey			Lent Survey		
	Positive Reactors	Contacts Examined	Average Child	Positive Reactors	Contacts Examined	Average Child
Traced to new cases	7	43	6·1	9*	49	5·4
Known cases not living in Bournemouth	9	50	5·5	48	129	3·0
Known cases in Bournemouth ..	17 already known			20 already known		
Untraced, but all available contacts examined	9	50	5·5	24	80	3·3
Untraced, but some children and contacts in this group never attended for mass radiography				34	—	—
Tuberculin reaction read as "doubtful"	0	—	—	28	—	—
Total	42	143		163	†	

* One of these children led to the discovery of two new cases of adult tuberculosis.

† In the second survey, inevitably, there was some failure to obtain full particulars in every case, and some defection of the contacts regarding attendance for mass radiography. (This was eliminated in the first survey by persistent effort and domiciliary visiting.) Altogether, 411 persons did attend the mass radiography unit, and 339 of these were the children and their contacts in the groups which were completely followed up. The remaining 72 persons were some of the tuberculin-positive children whose source of infection was not discovered and a few of their contacts. None of the children whose reaction was classed as "doubtful" was traced to a case of tuberculosis.

The following are particulars of the contacts of all positive reactors found in the Michaelmas survey, together with the new cases of adult tuberculosis found in the Lent term survey.

Traced to New Cases (Michaelmas Survey)

Case 1.—The child's *x*-ray film showed left hilar gland enlargement and collapse of the lingula. Mother and three lodgers normal. No other relations in Bournemouth. Father was a sergeant instructor in the Army. He was symptomless; but *x*-ray film revealed bilateral pulmonary tuberculosis, and he had a positive sputum.

Case 2.—The child had a primary lesion with atelectasis of left upper lobe. The mother's *x*-ray film showed a small cavity in the right upper lobe and she was sputum-positive. Six other contacts were normal.

Case 3.—Child and seven contacts normal. The father's *x*-ray film showed bilateral pulmonary tuberculosis and he was sputum-positive.

Case 4.—Child and five contacts normal. Mother had bilateral pulmonary tuberculosis and a positive sputum.

Case 5.—Child and four relatives in the house normal. A married sister in Reading was known to have pulmonary tuberculosis, and a friend of the family had been treated for pulmonary tuberculosis in a sanatorium. The father's *x*-ray film showed old-standing fibrotic disease in the left upper zone and mid-zone. Sputum and gastric lavage repeatedly negative.

Case 6.—Child and six contacts normal. Mother found to have a minimal lesion in the left upper lobe, sputum negative. Sister, a known case of pulmonary tuberculosis with a positive sputum, was living in the flat below in a converted house.

Case 7.—Child and four contacts normal. Father found to have a minimal lesion in his right upper lobe, sputum negative. A stepbrother, living in Blackpool, was a known case of open pulmonary tuberculosis; he had been in contact with the child prior to his diagnosis and treatment. The child stayed with him for holidays.

Traced to Known Cases (Michaelmas Survey)

Case 8.—Child and eight contacts normal. A known Bournemouth case of sputum-positive tuberculosis visited the house regularly.

Case 9.—Living in the same house with a known case of sputum-positive tuberculosis for three years. Two other children born in house after the man had died were both tuberculin-negative.

Case 10.—Child and five contacts normal. Mother a known case of sputum-positive tuberculosis, and a relation in the same house had recently had a pleural effusion.

Case 11.—Child and six contacts normal. Had previously been living with a known case of tuberculosis.

Case 12.—Lived with his aunt, who died three years ago of pulmonary tuberculosis. All contacts already examined.

Case 13.—Child and six contacts normal. A brother, with whom the child had been in contact, was in Manchester under treatment for tuberculosis.

Case 14.—Living in the same house as a known case of pulmonary tuberculosis. All contacts under supervision.

Case 15.—Child and four contacts normal. Two young people who previously lived in another flat in the same house were known to have had tuberculosis. One died a year ago of pulmonary tuberculosis, and another three years previously of tuberculous meningitis.

Case 16.—Child and four contacts normal. Father a known case of pulmonary tuberculosis under treatment in a sanatorium in America.

Case 17.—Child and four contacts normal. Close contact with a known case of pulmonary tuberculosis who died 18 months ago.

Case 18.—Child and five contacts normal. Recently moved to this country from China, where he was known to have been in contact with a case of tuberculosis.

In six other cases one of the parents had died of tuberculosis during the child's life, and all other contacts had been examined. In nine further cases the child had a tuberculous parent under supervision at the Bournemouth Chest Clinic, and all other contacts had been examined.

Source of Infection Untraced (Michaelmas Survey)

*Case A.**—This child's *x*-ray film showed a resolving primary lesion. Mother was normal. Four siblings were tuberculin-positive. One had segmental atelectasis, while another developed tuberculous meningitis 11 weeks after being tuberculin-tested. The father was dead, and, although several visitors to the household were examined, a lodger consistently refused examination.

Case B.—Child showed a primary lesion in the left lung. A like twin was found to be tuberculin-negative. The twin was observed over a period of four months and remained tuberculin-negative. Eight contacts were examined, all normal. They lived in a guest-house, and it seemed possible that they might have had unknown contact with a case of active tuberculosis.

Cases C and D.—Child and ten contacts normal.

Cases E and F.—Child and five contacts normal.

Case G.—Child and three contacts normal.

Cases H and I.*—Child and two contacts normal.

Traced to New Cases (Lent Survey)

Case J.—Child normal. Sister showed thickening of right interlobar septum, enlargement of hilar glands and atelectasis of right upper lobe; admitted to sanatorium. Brother, *x*-ray film normal. Mother, calcified foci right upper lobe. Father, infiltration and cavitation left upper lobe; sputum-positive. Sister, aged 9 months, found to be patch-positive; maintained under weekly observation and two weeks later developed tuberculous meningitis.

Case K.—Child had a primary lesion in the left upper lobe. Four contacts normal. Father had infiltration in right upper lobe; sputum-positive.

Case L.—Child and four contacts normal. Father had infiltration and cavitation in right upper lobe, sputum-positive.

Cases M and N.—Child and five contacts normal. Aunt and uncle, both living in the same house, found to have pulmonary tuberculosis. Uncle had infiltration and cavitation in both upper lobes; sputum-positive. Aunt had minimal lesion in right upper lobe; sputum and gastric lavage negative.

Case O.—Child and five contacts normal; lodger in same house had fibrosis in right upper lobe.

Case P.—Child and three contacts normal. Sister, aged 20, had a minimal lesion in her first left interspace.

Case Q.—Child had collapsed right middle lobe; three contacts normal. Mother had minimal lesions in her second left interspace.

Case R.—Child and two contacts normal. Father had infiltration in right upper lobe; sputum-negative.

Case S.—Child and five contacts normal. Father had a minimal lesion in the right upper lobe; sputum-negative.

Discussion

This investigation was possible only through close co-operation between the chest physicians and the medical officer of health. Whether diagnostic or preventive, it illustrates the importance of such collaboration in anti-tuberculosis work.

The results show that for every 250 tuberculin tests applied in the schools a new case of adult tuberculosis was discovered among the contacts. As mass tuberculin-testing can be carried out cheaply, this case-finding method is relatively inexpensive.

Only 411 persons passed through the mass radiography unit in the Lent survey, yet 10 new cases of adult tuberculosis were found. Such a group is obviously eminently suitable for mass radiography examination. However, in order to make use of the mass radiography unit in this way, it is necessary to carry out a large number of tuberculin tests in a relatively short time. In this particular investigation 411 examinees were easily dealt with by the unit in a fortnight in addition to other work, but it involved considerable rearrangement of the school nurses' programmes to enable them to apply and to read nearly 2,000 tuberculin tests during the same period. On the other hand, when the survey was protracted over a whole school term the tuberculin-testing was more easily arranged, but the examination of the contacts at the chest clinic involved some adjustment of the weekly clinic sessions there. It would seem that both procedures are equally satisfactory for finding cases, and the choice should depend on local facilities.

The particular value of this case-finding technique is that it "gets at" the unsuspected case by a "back-door" approach, and would seem to be especially suitable for an urban area where the milk had been extensively pasteurized during the past five years.

ADDENDUM.—Since this article was submitted for publication two more untraced cases in the Michaelmas survey have been traced to a source case.

Case A.—After prolonged and persistent efforts by the health visitor the lodger was persuaded to attend; the *x*-ray examination showed bilateral pulmonary tuberculosis, and he had a positive sputum.

Case H.—The mother of this child eventually attended, and the *x*-ray examination revealed a minimal lesion in the right upper lobe. Sputum was repeatedly negative.

This brings the total of new cases in the Michaelmas survey to nine and the total number of new cases discovered to 19.

Summary

Two tuberculin surveys of infant-school children were conducted with the object of discovering adult tuberculosis among the household contacts of the tuberculin-positive children.

In the Michaelmas survey, 1,865 children were tuberculin tested by a chest physician and the contacts of positive reactors examined by fluoroscopy. In the Lent survey, 1,933 children were tuberculin-tested by school nurses and the contacts of positive reactors examined by mass radiography. In both surveys one new case of adult tuberculosis was found among the contacts for every 250 tuberculin tests applied in the schools. The co-operation of the mothers of the children in persuading the contacts to attend is regarded as a most important detail in the scheme.

This would appear to be a cheap and effective case-finding method, especially suitable in an urban area where the milk is pasteurized.

Our thanks are due to Dr. J. S. Robertson, medical director of the mass radiography unit; Mr. Childs, the education officer for Bournemouth; the Primary Education Committee for permission to carry out this investigation; and also the head teachers and staffs of the schools concerned and the staff of the health department and chest clinic.

*See Addendum.

REFERENCES

Davies, T. E. Jones (1950). *Brit. J. Tuberc.*, **44**, 1.
Dick, W. P. (1950). *British Medical Journal*, **2**, 141.
Hall, A. S., and Tattersall, W. H. (1950). *Lancet*, **1**, 490.
MacDougall, I. A., Mikhail, J. R., and Tattersall, W. H. (1951). *Med Offr.*, **86**, 253.
Medical Research Council National Tuberculin Survey (1952). *Lancet*, **1**, 775.
Tattersall, W. H. (1946). "Abstracts" of the British Tuberculosis Association, March 15, 1946.
Wolman, B. (1952). *Brit. J. Tuberc.*, **46**, 24.

THE ACUTE CHEST IN GENERAL PRACTICE

BY

JOHN FRY, M.B., F.R.C.S.

Respiratory disorders comprised 12% of all attendances in a general practice over a period of one year (Fry, 1952). Together with the upper respiratory infections the respiratory tract accounted for over 25% of all work done in this particular practice. Admittedly, a very large proportion of these ailments were of a mild and transient nature producing no detectable changes in the lungs on clinical examination. Excluding these minor cases, there remain two large groups of more serious chest disease—the acute and chronic disorders.

It is with the former that this paper is concerned. "The acute chest" is a useful term to denote sudden severe chest disease producing definite clinical signs of respiratory tract involvement.

This report is on a series of 164 patients with this type of disorder. They were seen over a period of two years (July, 1950, until July, 1952) in a general practice of over 4,000 patients on the outskirts of London. During this same period 15 new cases of pulmonary tuberculosis were diagnosed, 800 patients were treated for short-term coughs with no chest signs, and 55 patients required repeated treatment for their "chronic bronchitis." This area is not regarded as presenting any extra hazards so far as chest diseases are concerned.

A special effort was made to obtain extra information from radiological and pathological investigations in addition to careful clinical records, but anyone who has had close association with general practice will at once understand that this ideal is a practical impossibility in a number of cases. Thus, not all the cases have been fully investigated according to hospital standards.

The cases are best considered under the following accepted clinical groups: acute bronchitis, the pneumonias, acute pleurisy, severe asthma, and spontaneous pneumothorax.

Acute Bronchitis

This term is rather a vague one. Different interpretations of it are given by different practitioners. Here, it is taken to describe a moderately severe chest illness of sudden onset and varying constitutional upset with bilateral and diffuse clinical signs of involvement of the respiratory tract. It should be stressed that it affects more or less the whole of the "middle respiratory tract" of both lungs.

In this series two subgroups were noted—a primary group, in which the illness occurred in persons with no previous history of chest disease, and a secondary group in which the acute attack complicated some active or latent chest condition.

It is doubtful whether acute bronchitis is in fact ever a primary condition in the true sense of the term, as it is invariably a downward extension of a preceding upper-respiratory-tract infection. The reasons why in these instances this downward spread occurs are not obvious, nor is it clear why the condition should stop short and affect only the bronchi and not proceed to cause definite parenchymal involvement. In fact, it has been found most difficult to differentiate clinically between severe acute bronchitis and so-called bronchopneumonia. The usual differentiation seems to be based on the degree of severity of the illness and on whether rales (which are taken to denote parenchymal involvement) are present in addition to rhonchi.

It is probable that acute bronchitis occurs when the defence mechanisms of the respiratory tract have been rendered ineffectual either by a preceding disease such as "chronic bronchitis," or when, for some reason, it follows on an upper-respiratory-tract infection.

The actual organisms which are detected from the sputum are non-specific, and in this series consisted of the normal inhabitants of the upper respiratory tract (*Neisseria catarrhalis* and *Streptococcus viridans*), with the occasional addition of pneumococci and haemolytic streptococci.

Sixty-six patients were treated for one or more attacks of acute bronchitis in the past two years. The group distribution was as follows.

Primary Group.—This consists of 23 patients (10 males, 13 females). The age distribution is as shown in Table I. Nineteen cases occurred in the winter and spring months.

Secondary Group.—This consists of 43 patients (18 males, 25 females), with 51 actual attacks. Again all but five occurred in the winter and spring. The age distribution was different from that of the primary group in that this condition is more frequent in the older age groups. The associated lung disorders were: asthma in 12 patients (7 children), bronchiectasis in 2, "chronic bronchitis" in 23, and measles in 6 (it was considered that the bronchitis in measles was a secondary form).

TABLE I

Group	Age Group in Years								
	0–4	5–9	10–19	20–29	30–39	40–49	50–59	60–69	70 and Over
Primary	8	4	—	—	1	2	1	4	3
Secondary	3	10	1	1	2	4	3	8	11

Clinical Features

The actual clinical features in both groups were alike.

Onset.—This was more sudden in the primary group, with its preceding upper-respiratory-tract infection. Rapid progression with increasing severity of the disease is particularly likely to occur in young infants, as was seen in two patients in this series, both of whom were under 6 months old.

Symptoms varied in degree. There was a dry, irritating, and distressing cough which became productive after a few days. No chest pain occurred, apart from some retrosternal discomfort. Dyspnoea was often present.

Signs.—The pulse rate was always raised; the fever varied from 99 to 104° F. (37.2 to 40° C.), being least marked in the aged, although there was often pronounced tachycardia. The respiratory rate was usually increased. Examination of the chest revealed diminished chest movement, normal resonance on percussion and on auscultation, numerous bilateral and widespread rhonchi, with an especially prolonged expiratory phase in the secondary group. The presence of associated cardiac failure in the aged was a common finding.

X-ray examination was possible in four patients in the acute attack, increased pulmonary striations with some possible consolidation being noted at both bases in one case. No significant changes were present in the others.

Sputum was examined in 40 patients, and the common organisms found were *N. catarrhalis, Str. viridans,* pneumococci, haemolytic streptococci, and occasionally *Staphylococcus aureus.*

Treatment consisted of: (1) Treating the infective process with parenteral procaine penicillin in the more severe type of case, the adult dosage being 700,000 units daily in single doses. The response was satisfactory in most cases, particularly in the primary group. Those in the secondary group which did not respond to penicillin were given chloramphenicol, 500 mg. six-hourly (adult dose), with no further beneficial effects. (2) The use of sedatives to ensure adequate rest was regarded as important. (3) Where bronchospasm was marked, the use of antispasmodics such as adrenaline, ephedrine, and isoprenaline sulphate was of some value.

Course.—All but one patient recovered; the time for recovery was from 6 to 28 days, being most rapid in the primary group. Three patients were admitted to hospital: (1) An infant aged 3 months who was very ill and whose home facilities were inadequate. (2) A woman aged 36 with recurrent asthmatic attacks, who developed a tension pneumothorax during an attack of acute bronchitis. (3) A man aged 57 with 30 years' history of progressively more severe chronic bronchitis; he developed severe cardiac failure during an acute attack, was admitted to hospital, and died six weeks later.

The Pneumonias

Pneumonia is here taken to mean an acute illness with clinical signs of involvement of the lung tissues—it is in fact an inflammation of the lung with alveolar exudate as shown by clinical, and in some cases additional radiological, findings. As with other types of acute respiratory disorders, the aetiology and classification of this condition are far from satisfactory—we have a number of terms purporting to describe different syndromes. These terms may be based on aetiological concepts: we have specific—for example, pneumococcal, streptococcal, etc.—and aspiration pneumonias; on morbid anatomical grounds we have lobar and lobular pneumonias; and topographically there are such subdivisions as massive, central, apical, and creeping varieties. In more recent times we have had the addition of "primary atypical" and "virus" pneumonias. All this is rather confusing, especially so to the general practitioner, who relies largely on clinical findings and who now treats the majority of pneumonias without reference to hospital (in this series only one out of the 77 cases under consideration was sent to hospital).

It is not the purpose of this paper to attempt a reclassification, but a plea is made that appropriate investigations be undertaken to clarify the whole field of acute lung infections as well as to study further the functions and defence mechanisms of the respiratory tract as a whole. A step in this direction has been made by Scadding (1951), who suggests an aetiological basis for classification of pneumonias.

A relatively simple method has been used in this instance, and grouping has been on clinical and possible aetiological grounds (admittedly this is not entirely satisfactory).

Group 1. Cases with Lung Consolidation (7 Cases)

In this group are those cases of what is now termed "classical pneumonia." It is an illness of sudden onset with severe constitutional disturbance and clinical signs of lobar consolidation. In the experience of this practice it is now a relatively rare disease. Only seven cases were seen over the period in question (10% of all pneumonias), and four of these were complications of influenza. It is striking that all patients were over 55 years of age, three being over 70. All were very ill, with sudden onset, high fever, tachycardia, and dyspnoea. Locally, evidence of consolidation was found in all cases. Response to penicillin (700,000 units daily) was rapid, and complete recovery occurred in two to four weeks. Sputum contained pneumococci in two, haemolytic streptococci in two, and *Staph. aureus* in one case. One patient was admitted to hospital on account of unsatisfactory home arrangements. There were no deaths.

Bronchopneumonia is a common and favourite diagnosis, and, as has already been mentioned, it is differentiated from bronchitis by the severity of the illness and the presence of signs denoting involvement of the lung parenchyma. Great difficulty was experienced in making this differentiation, and rather than attempt this distinction all such cases were diagnosed as acute bronchitis and dealt with in that section.

Group 2. Localized Pneumonia with no Clinical Signs of Consolidation (70 Cases)

The most common form of pneumonia encountered was a relatively mild condition producing identical signs in all cases, but in which there was a possibility of more than one aetiological process. This syndrome presented as a fairly sudden illness in a fit person, with a distressing cough, little real constitutional upset, and characteristic signs on auscultation of the chest consisting of diminished air entry and a shower of rales at the end of inspiration. An attempt was made to group these cases in three possible aetiological sections.

A. Probable Viral Influence (35 Cases).

Thirty-three such cases were seen in the influenza epidemic of 1950–51 (Fry, 1951). Although no conclusive tests were carried out to prove the viral aetiology, these cases occurred during a nation-wide epidemic which other observers had shown to be due to influenza A virus. The onset of lung involvement in the majority occurred a few days after the beginning of influenzal symptoms, the general picture being as already described. One or other base was involved in 30 instances, and the right upper zone in three. A pleural rub was heard in three patients. *Sputum* was mucopurulent, blood-staining occurred in two cases, and bacteriological examination showed the presence of *Str. viridans, N. catarrhalis,* and haemolytic streptococci in the six cases so investigated. There were no abnormal radiological findings in the four patients *x*-rayed. There was no definite response to antibiotics, but recovery was eventually complete in all cases, taking from 2 to 28 days for signs to clear. Since the end of 1951 all such cases were investigated by virus complement-fixation tests, but there has been no epidemic, and only one case of influenza B with identical clinical findings was seen in March, 1952. In June, 1952, a case of probable psittacosis was seen. This is described because of its relatively mild nature.

A normal healthy schoolgirl aged 11 was seen at home on June 23 with a history of malaise, fever, and cough for one week. She did not appear ill or distressed apart from an irritating dry cough. The pulse rate was 70 and temperature 99° F. (37.2° C.). Examination of the chest revealed diminished movement and dullness at the left base, with inspiratory rales and diminished air entry. A diagnosis of pneumonia was made and she was given 500,000 units of procaine penicillin intramuscularly daily for the next five days. As she was not really ill no dramatic improvement occurred and the signs cleared slowly. By July 7 the chest was free of abnormal signs. The results of investigations were as follows. On June 23 the white cell count totalled 6,500 and the E.S.R. was 40 mm. in one hour. A radiograph taken at the patient's home showed "extensive consolidation of basal segments of the left lower lobe with some associated collapse" (Dr. P. S. G. Campbell). On July 17 her chest *x*-ray film was clear. Blood was sent to the Virus Reference Laboratory (on the ninth day of illness), and the report was that the serum reacted to a titre of 1/160 against the psittacosis group of viruses. This was thought to indicate a specific infection. Serum on the eighteenth day of disease reacted against a titre of 1/80. Frei's test on July 4 was negative. Questioning revealed that the patient had played with a bird's nest one week before the onset of illness. No other contact with birds was elicited.

B. Probable Aspiration Pneumonia (21 Cases).

In these cases infected mucus from the upper respiratory tract, acting as a bronchial embolus, producing an area of collapse and infection, was considered the probable cause. In 18 cases there was a definite preceding history of an acute upper-respiratory-tract infection, and in three there was a recurring catarrhal condition causing a persistent cough, but with no organic changes in the lungs. The age distribution is shown in Table II. It will be seen that most cases occurred in the under 10 group, and those were inclined to be "catarrhal children." Twelve cases occurred in the winter months, 8 in the summer, and one in the autumn. There were 10 males and 11 females. The areas of lung affected were right base in 9, left base in 10, right upper zone in 1, and left mid-zone in 1.

TABLE II

Age:	0–4	5–9	10–19	20–29	30–39	40–49	50–59	60–69	70 and Over
No. of cases..	3	8	2	—	3	4	—	1	—

The condition presented with a gradual onset (although on four occasions it was sudden) following an upper-respiratory-tract infection. The chief complaint was the irritating cough and some degree of general malaise. No patient was considered to be very ill. Temperature varied from normal to 102° F. (38.9° C.) and there was a relative bradycardia. The signs were identical with those described in the preceding group. These signs cleared in from four days to two months, the average being twelve days.

Treatment did not appear to influence the course of the disease except where there was more pronounced constitutional upset; then penicillin was given and the toxaemia was rapidly controlled. The white-cell counts varied from 25,000 to 5,000 (the average of eight cases being 8,000), and the average E.S.R. figure (for six cases) was 10 mm. in one hour. Only *N. catarrhalis* and *Str. viridans* were grown from six sputa. All virus complement-fixation tests were negative (five cases), cold agglutinins were present in one case out of eight tested. Radiographs were taken in 17 cases either at home or at hospital. In 12 of these some abnormality was detected. This abnormality consisted of small dense patches in the affected areas. Associated collapse was reported in four children. Hilar glands were enlarged in three instances.

C. Possible Specific Cause (at present unidentified) (14 Cases)

The clinical picture was identical with that of the previous group with the exception of a really sudden onset, no preceding catarrhal condition of the upper respiratory tract, and more severe constitutional reaction. There were seven males and seven females. The age incidence is shown in Table III.

TABLE III

Age:	0–4	5–9	10–19	20–29	30–39	40–49	50–59	60–69	70 and Over
No. of cases..	—	5	3	1	1	2	—	1	1

The signs cleared in 4 to 21 days, with an average of 7.5 days. In 10 cases the right base was affected, in four the left base, and in one the left mid-zone. *X*-ray findings showed abnormalities in three out of seven cases examined. A more diffuse opacity was observed. Sputa from seven cases were found to contain the usual organisms of the upper respiratory tract, but in three cases *Staph. aureus* was found and in one haemolytic streptococci. The white-cell counts varied from 5,000 to 13,000 (average 9,000). Virus complement-fixation tests were negative in six cases. Cold agglutinins were also absent. Three cases were treated with aureomycin, 500 mg. six-hourly, but in only one was a dramatic change noted. Penicillin in six cases was again found to control the systemic upset, but signs persisted. The remainder were treated symptomatically. There was no mortality and no patient was admitted to hospital.

Differential Diagnosis

The question of whether these conditions might be due to any other specific state must be considered. The possibilities which arise are carcinoma of the bronchus causing bronchial obstruction with collapse and inflammation of the affected lung segment. Follow-up in this series has excluded this possibility. Pulmonary tuberculosis of the progressive type was also excluded by follow-up. Tuberculin jelly skin tests were carried out on all the children and were negative (two positive reactors with similar signs were excluded from this report). Loeffler's syndrome has also been considered, but in the 11 patients who had a differential count there was no evidence of any eosinophilia.

Comment on the Pneumonias

Some would place these pneumonias in the so-called "primary atypical" group. This term, however, is a most unfortunate one, as it implies some standard condition which must be regarded as a typical pneumonia and also that the condition is due to a specific agent. A much more reasonable explanation is that this type of condition can be brought about in a number of different ways. Is it in fact a new disease, or was it recognized in the past and known as "congestion of the lung" or "bronchial catarrh"? It is being seen more frequently in hospital practice. Reimann (1943) stated that these conditions account for 70% of all pneumonias; Scadding (1951) showed how the incidence of pneumococcal pneumonia had dropped from 70 to 40% of all pneumonias, and that in 1949–50, at the Hammersmith Hospital, 20% of pneumonias were due to viruses, and in 23% the aetiology was inconclusive. In my series this group accounted for 90% of cases.

As has already been stated, there are probably multiple causes for this syndrome. It has already been proved that *viruses*—for example, psittacosis and rickettsia—can cause this type of condition. Influenza presents a more difficult problem. In fatal cases of influenza with lung involvement secondary infection with bacteria is usual, and in "influenzal pneumonias" sputum culture usually grows organisms which might be pathogenic. In addition, influenza often begins as a catarrhal condition of the nose and throat; and it is possible that this catarrh might produce bronchial embolism. Therefore it is doubtful whether the influenza virus is the only factor concerned. A most important possibility is that of *bronchial embolism* by mucus from catarrhal conditions of the upper respiratory tract. Animal experiments have shown that the introduction of viscid mucus into the trachea is followed by collapse and infection of areas of the lungs. Quinn and Meyer (1929) showed that if radio-opaque oil is placed in the nose of a normal sleeping subject this oil will be found in the lungs the following morning.

It was Brock (1946) who stressed the great importance of bronchial embolism and gravitation in the causation of lung abscess. Nicholson (1950) put forward the view that bronchial embolism accounted for a whole range of conditions, from benign aspiration pneumonia (similar to cases described above) through an intermediate non-specific suppurative form to acute lung abscess. Lees (1950) described pulmonary lesions resulting from probable blockage of the bronchi by tenacious mucus in whooping-cough.

Ramsay and Scadding (1939) described a series of 21 cases with radiological changes which were seen at a chest clinic, and they ascribed these to a similar cause. Scadding (1948, 1951) and Robertson and Morle (1951) have attempted to show that the "primary atypical pneumonia" syndrome is in fact an aspiration pneumonia. This particular series includes 35 cases with a probable viral influence, 21 which were probably aspiration pneumonias, and 14 which may have had a specific cause, either viral or bacterial, that at present is unidentifiable.

Acute Pleurisy

In the period under discussion six cases (three male and three female) of apparent primary pleurisy were seen in which, apart from a pleural rub at the corresponding painful region, there were no other abnormal chest signs. The patients' ages ranged from 20 to 75, but four were under 40. Five were *x*-rayed in the acute phase, and in all a small patch of consolidation was found in the affected area. On follow-up this cleared within two weeks, with the exception of one case in which it persisted for two months. Follow-up did not reveal any tuberculous process in any of these patients. The question arises whether pleurisy is ever a primary condition and whether it is always secondary to some lung lesion. In this same period three cases of acute tuberculous pleurisy were seen in young adults.

Severe Asthma

This accounted for 10 cases of "acute chest." It produced marked distress in the patient and gave great anxiety to both relatives and medical adviser. The sex distribution was equal and the age incidence varied from 10 to 65. Two patients required treatment in hospital.

Spontaneous Pneumothorax

This is included as it accounted for five cases. It had a sudden onset with pain and a variable degree of dyspnoea. In four there was no previous or subsequent chest trouble. The fifth patient, an elderly lady of 68, had a history of recurring coughs and developed a valvular pneumothorax. She died in hospital.

Conclusion

It will be seen that these numerous cases from a single general practice present a different picture from those seen in hospital work. This is, of course, due to the fact that only the cases which cannot be managed at home are sent into the hospital wards. This series was relatively benign—thus only two patients died in this group and nine required admission to hospital. This type of case has received almost no attention in the past, and it is felt that there is a need for further study of this group. A simpler and more precise method of classifying acute chest diseases, which should be based on aetiological grounds, is also necessary.

Summary

A clinical report is presented on 164 cases of acute chest disease as seen in a general practice over a period of two years.

Acute bronchitis accounted for 66 cases. Subdivision into primary and secondary groups is made.

Seventy-seven cases of pneumonia were seen. Only three of these were the primary "classical" pneumonias. The common type was a localized area of inflammation with no clinical evidence of consolidation. On the aetiology of this subgroup it is difficult to dogmatize, but three possibilities are put forward: (1) infection due to an identifiable virus; (2) an infection due to a specific unidentifiable agent; or (3) the condition might in fact be an aspiration pneumonia.

The other conditions which produced the acute chest were pleurisy, severe asthma, and spontaneous pneumothorax.

A plea is made for a simpler and more logical classification of acute chest diseases, and the need for further research on these conditions is stressed.

I wish to express my gratitude to the pathological and radiological departments of the local hospital for their assistance, especially Dr. A. C. Glendinning and Dr. John Keall, and also to the Virus Reference Laboratory, Colindale.

REFERENCES

Brock, R. C. (1946). *Anatomy of the Bronchial Tree*. Oxford Univ. Press, London.
Fry, J. (1951). *British Medical Journal*, 2, 1374.
—— (1952). Ibid., 2, 249.
Lees, A. W. (1950). Ibid., 2, 1138.
Nicholson, H. (1950). *Lancet*, 2, 549, 605.
Quinn, L. H., and Meyer, O. O. (1929). *Arch. Otolaryng., Chicago*, 10, 152.
Ramsay, H., and Scadding, J. G. (1939). *Quart. J. Med.*, 8, 79.
Reimann, H. A. (1943). *Bull. N.Y. Acad. Med.*, 19, 177.
Robertson, P. W., and Morle, K. D. F. (1951). *British Medical Journal*, 2, 994.
Scadding, J. G. (1948). *Lancet*, 1, 89.
—— (1951). *Proc. roy. Soc. Med.*, 44, 511.

A radical change in the footwear of this year's British Everest expedition is announced. Instead of the familiar heavily nailed leather climbing-boots, the assault parties will wear a much lighter boot with soles of artificial rubber and glacé kid uppers. The boots are insulated inside with a layer of kapok nearly an inch thick. It is pointed out that lightness and warmth are far more important qualities than durability and water-resistance for the Everest assault. The boots, which have already been tried in the Alps, will be required only for a few days' use, and at the temperatures high on Everest water is unlikely to be encountered (*The Times*, January 6).

DISSECTING AORTIC ANEURYSM: A VARIED CLINICAL PICTURE

BY

R. K. MacCUISH, M.B., M.R.C.P.

Senior Medical Registrar, Thoracic Surgical Centre, Shotley Bridge Hospital, Co. Durham

Dissecting aneurysm was first described by Morgagni in 1761. In 1934 Shennan published his exhaustive monograph on the subject, having been able to collect over 300 cases from the literature. A significant point arising from this report was that only six cases were diagnosed before death.

The correct ante-mortem diagnosis may present much difficulty for two reasons. (1) Bizarre symptoms and signs may occur, and disease of other systems may be simulated. (2) It is a somewhat uncommon condition in hospital practice. Many cases die suddenly and therefore fall into the hands of the coroner. In San Francisco, Mote and Carr (1942) compared the incidence at coroners' post-mortem examinations with that at hospital post-mortem examinations. They found three times as many in the former as in the latter, and a similar state of affairs may well hold in this country.

The clinical picture which has come to be recognized as typical of aortic dissection is that of the sudden onset of severe tearing thoracic pain accompanied by shock and radiating down both legs. This view of dissecting aortic aneurysm may have obscured the correct diagnosis in many instances in the past, and patients may have been subjected to operative intervention when this condition was not borne in mind. It is the purpose of this note to suggest that aortic dissection may often be a silent process and may progress with only minor symptoms for months or years.

Five cases are described, illustrating the paucity of symptoms and the tendency to simulate other diseases.

Case 1

A 53-year-old woman was admitted to hospital with 30 years' history of epigastric pain relieved by food and alkaline powders. For two to three years previously the pain had radiated through to the back. Two weeks before admission she developed a sudden weakness in the left arm and left leg. Twelve hours before admission she had a severe attack of epigastric pain radiating through to the back. She persisted that the sudden pain which occurred before admission was of the same nature as that which she attributed to her dyspepsia.

On examination her general condition was reasonably good. The pulse was regular and the blood pressure 230/130. The apex beat was not displaced. A systolic murmur was present over the praecordium. There was definite muscular weakness of the left arm and leg, with exaggerated reflexes and a doubtful plantar response. There was no abnormal finding in any other system.

Investigation.—Haemoglobin, 56%; white cells, 15,000 (polymorph leucocytosis). Urine: albumin +, granular and hyaline casts, red blood cells present. The blood urea was 70 mg. per 100 ml. The Wassermann reaction was negative. An electrocardiogram showed inversion of T_1, T_2, and T CR 4.

The epigastric pain persisted, resisting all the usual methods of treatment. Strong sedation had to be employed. Five days after admission a small effusion was detected clinically at the left lung base, and this extended up to the spine of the scapula during the next 48 hours. *X*-ray examination of the chest revealed almost complete opacity of the left hemithorax. Repeated attempts

at aspiration gave only a few drops of blood-stained fluid. The patient went slowly downhill and died 17 days after admission.

Necropsy.—Hypertrophy of the left ventricle and a marked atheroma of the aorta were found. There was a massive effusion of clotted blood in the left hemithorax. A dissecting aneurysm began in the ascending arch of the aorta and extended down to the renal arteries. In the thorax the aneurysm had developed a secondary fusiform swelling, the wall of which was composed of old laminated film clot. This had ruptured, to give rise to the haemothorax. There was no evidence of peptic ulcer.

Case 2

A 71-year-old night watchman was admitted with two years' history of intermittent attacks of left-sided abdominal pain. It sometimes radiated round to the lumbar region and to his back, but was never severe enough to make him stay away from his work. On the day of admission the pain was so severe that he collapsed and was brought to hospital.

On examination his general condition was reasonably good; blood pressure, 200/120. There were no abnormal physical signs. The urine showed: albumin + +, many red blood cells, no casts. The blood urea was 180 mg. per 100 ml. A radiograph of the chest showed dilatation of the aortic knob, suggesting an aneurysm. *X*-ray examination of the abdomen and spine showed nothing abnormal.

The pain persisted and at times was severe enough to necessitate strong sedation. The day after admission he passed only 6 oz. (170 ml.) of urine, and on the second day he became anuric. Cystoscopy was performed. The bladder and both ureteric orifices were normal. Ureteric catheters could not be passed. The left kidney was therefore explored. It was found to be swollen and pale, and the surface was stippled with multiple infarcts. No urine was present in the renal pelvis. The patient died without recovering consciousness.

Necropsy.—The aorta was grossly atheromatous. A dissecting aneurysm was present, beginning below the renal arteries and extending up to the aortic arch, where it stopped short. This occluded the renal arteries. Both kidneys were completely infarcted. There was pronounced hypertrophy of the left ventricle.

Case 3

A 60-year-old woman was admitted with a history of progressive dyspnoea on exertion for nine years and swelling of her feet for two years. For four months she had experienced a heavy substernal pain, not particularly related to exertion or to food. It sometimes lasted up to 30 minutes, but was never unduly severe.

Examination showed the patient to be very obese and obviously myxoedematous. She was dyspnoeic and cyanosed at rest. Her blood pressure was 190/110; pulse regular; apex beat displaced to the left. The heart sounds were normal. Oedema of feet was present and there were rales at both lung bases. The urine showed: albumin + +, many hyaline casts, no red blood cells. The blood urea was 58 mg. per 100 ml. An electrocardiogram showed RS–T depression in Leads I, II, and III, and T inversion in CR 4 and CR 7.

On the day after admission she suddenly collapsed and became deeply cyanosed, with gasping respirations, sweating, and bounding pulse. She slowly recovered consciousness, and during the following week became less dyspnoeic and cyanosed. At the end of the first week she had another attack similar to that described above, and died soon afterwards.

Necropsy.—The aorta was grossly atheromatous. A dissecting aneurysm was found, beginning in the aortic arch and extending to just above the origin of the renal arteries. The left ventricle was hypertrophied. The thyroid was small and atrophic. There were no other gross pathological findings.

Case 4

A 48-year-old police officer suddenly lost consciousness while waiting in a dentist's surgery. He was taken to hospital, and on admission was violent and uncontrollable, and threw himself around on the receiving-room floor. After three hours he recovered consciousness fully. His blood pressure was 90/60. The lips were very cyanosed. There was obvious weakness of the left arm and leg. The left arm was cold and discoloured and no arterial pulsation could be felt. The heart was fibrillating. The reflexes were somewhat increased in the left arm and leg, and the left plantar response was extensor. The patient died suddenly the next day.

Necropsy.—There was a large dissecting aneurysm, beginning ½ in. (1.3 cm.) above the aortic valve and extending as far as the coeliac axis, surrounding the innominate and left common carotid vessels, and effusing extensively along the left subclavian artery. It had ruptured into the pericardium and extended around both lung roots. There was considerable hypertrophy of the left ventricle.

Case 5

A 53-year-old railway signalman was admitted to hospital with a history that four weeks previously, while standing in the street, he had a sudden severe pain in the chest radiating to both groins. This pain passed off in a minute and left his right leg numb and weak. The whole condition passed off in 15 minutes, and nothing like it recurred up to his admission to hospital.

On examination his blood pressure was 260/170. His heart was not clinically enlarged. A soft diastolic murmur was heard at the left sternal border. There were no other abnormal clinical findings. The urine showed: albumin + + and excess of leucocytes in deposit. The blood urea was 66 mg. per 100 ml. The Wassermann reaction was negative. A blood count showed: Hb, 12.3 g.%; white cells, 10,500. Cardioscopy revealed slight enlargement of the left ventricle. An electrocardiogram showed left ventricular hypertrophy.

A course of oral hexamethonium bromide was begun 10 days after admission, but 12 days later the patient awoke during the night complaining of upper abdominal pain and collapsed. He was found to be comatose and sweating profusely, and his blood pressure fell to 80/60. Consciousness was recovered fairly quickly, but two similar episodes followed at three-day intervals. After his first attack he had a tender swelling in the right loin which progressively increased in size. The blood urea rose to 480 mg. per 100 ml. He slowly became comatose, and died one month after admission to hospital.

Necropsy.—A dissecting aortic aneurysm was found, beginning in the arch of the aorta and tracking down the vessel beyond the origin of the renal arteries. There was a large retroperitoneal haemorrhage on the right side, surrounding the right kidney and extending from the lower edge of the right lobe of the liver into the pelvis. Both kidneys showed the changes of pyelonephritis. The heart was generally enlarged, with hypertrophy and dilatation of the left ventricle.

Discussion

Age and Sex Incidence.—No age group is free from this disease. The fifth and sixth decades claim the largest proportion of subjects, but a not inconsiderable number fall into the earlier periods. One of the youngest patients was a 22-year-old hypertensive male in whom multiple tears were found after death (Flaxman, 1942). Some indication of the incidence in the younger age groups is given by Schnitker and Bayer (1944), who found that 24% of their series were under 40. Males are subject to this disease more than females. Baer and Goldburgh (1948) suggest that as many as two-thirds of all cases occur in males.

Pathology.—It was originally believed that atheromatous degeneration was responsible for the initiation of dissection.

Shennan (1934), however, pointed out that most dissections begin in areas relatively free from atheroma. Atheromatous degeneration is seldom very severe in the first part of the aorta, where dissection most often begins. Cystic degeneration of the media, frequently found in aortic dissection, is now generally accepted as the initial lesion. Some writers have stressed the importance of sudden strain in initiating the tear, but such a history is often absent. In none of the above five cases was sudden strain an important factor.

Clinical Picture

A wide diversity in the presenting symptoms is manifest in these cases, ranging from complete absence of pain, as in Case 4, to the classical picture of severe chest pain radiating to the groin in Case 5. In the latter, however, the symptoms were of momentary duration only.

Silent dissection may not be so uncommon. Baer and Goldburgh (1948), in reviewing a series of 44 cases, found 24 which gave no history whatever of pain. There does not seem to be any very obvious reason why some cases should be painful. It may be that rapidity of dissection is a related point. In Case 4, in which there was no history of pain, the post-mortem findings pointed to a recent dissection, and from a clinical aspect dissection had extended from the origin of the left subclavian artery to the coeliac axis in 24 hours—that is, the time between the onset of symptoms and death.

The experience of these cases certainly does not support the view of Paullin and James (1948), who consider that pain may be absent when dissection proceeds slowly. In Cases 1, 2, and 3 pain was a prominent feature in the history, yet the pathological findings pointed to chronic dissection. Cases 1 and 2 are instructive in demonstrating how slight may be the incapacity resulting from chronic dissection. In Case 1, in view of the absence of peptic ulceration at necropsy, it is most probable that symptoms in the later two to three years of life were due to the aortic disease, yet her disability was never severe enough to warrant hospital admission up to the time of her last illness. Case 2 made light of his two-years history of left-sided abdominal pain, yet there can be no question that his symptoms were due to the dissection of the aorta.

That dissecting aneurysm of the aorta can simulate primary disease in other systems is readily understood on anatomical grounds. Extensive dissection to the carotid vessels may show as cerebral symptoms, to the subclavian arteries as vascular lesions in the arms, to the coeliac axis and renal vessels as abdominal emergencies or renal disease respectively. When, in addition, pain is minimal or absent the clinical diagnosis may become still more obscure. Cases 2 and 5 bear this out.

Renal involvement was the presenting feature in Case 2 and a later manifestation in Case 5. Blain, Glynn, and Hiratzka (1945) have reviewed the literature up to that time bearing on the question of renal involvement in aortic dissection.

From the neurological aspect Weisman and Adams (1944) have commented on the frequency of neurological symptoms in this disease. They mention that 30% of such cases coming to necropsy at the Boston City Hospital had such symptoms. In addition the combination of neurological and vascular lesions in a limb in any patient should immediately arouse the suspicion of aortic dissection.

Electrocardiographic Findings.—These are not infrequently a further source of confusion in reaching a diagnosis. The coronary blood flow is said to be obstructed by extension of the dissection to the coronary ostia or by compression of the vessels from pericardial haemorrhage, thus giving rise to the electrocardiographic picture of cardiac infarction. Bourne and Mills (1946) have reported two cases in which neither of the above explanations sufficed to explain the electrocardiographic findings. There the serial changes compatible with cardiac infarction were found in life, but at necropsy there was no evidence of cardiac infarction. Oram and Holt (1950) have reviewed the literature on this aspect, and suggested some features which might be helpful in the differential diagnosis.

Summary

Five cases of dissecting aneurysm of the aorta are described. It is suggested that less importance should be attached to the history of severe thoracic pain than in the past. Aortic dissection may often be a silent process or present with the minimum of symptoms over a prolonged period. The tendency to simulate other diseases is a prominent feature.

I am indebted to Dr. J. K. Rennie, Bradford Royal Infirmary, for permission to publish Cases 1–4, to Dr. W. G. A. Swan, Newcastle General Hospital, for Case 5, to Mr. C. L. O. Macalister for his notes of the cystoscopy and operation on Case 2, and to Drs. R. Turner and B. Smith for access to their necropsy reports.

References

Baer, S., and Goldburgh, H. L. (1948). *Amer. Heart J.*, 35, 198.
Blain, A., Glynn, T. P., and Hiratzka, T. (1945). *J. Urol.*, 53, 753.
Bourne, G., and Mills, P. J. W. (1946). *Brit. Heart J.*, 8, 181.
Flaxman, N. (1942). *Amer. Heart J.*, 24, 654.
Morgagni, G. B. (1761). *De sedibus et causis morborum*, 26, art. 21.
Mote, C. D., and Carr, J. L. (1942). *Amer. Heart J.*, 24, 69.
Oram, S., and Holt, M. C. (1950). *Brit. Heart J.*, 12, 10.
Paullin, J. E., and James, D. F. (1948). *Postgrad. Med.*, 4, 291.
Schnitker, M. A. and Bayer, C. A. (1944). *Ann. intern. Med.*, 20, 486.
Shennan, T. (1934). *Spec. Rep. Ser. med. Res. Coun., Lond.*, No. 193.
Weisman, A. D., and Adams, R. D. (1944). *Brain*, 67, 69.

LEAKING ABDOMINAL ANEURYSMS

TWO UNUSUAL CASES

BY

J. WOODROW BETTS, F.R.C.S.

AND

B. C. ROWLANDS, F.R.C.S.

Surgical First Assistants, the Royal Infirmary, Sheffield

Aneurysm of the abdominal aorta is an uncommon condition whose mode of presentation is unusually varied. The two cases presented here are recorded for their similar and misleading clinical features. They presented as strangulated inguinal herniae.

The importance of recognizing that aortic aneurysm may present as an acute abdominal emergency lies in the fact that unnecessary operation may thereby be obviated. Both the ruptured and the unruptured aneurysm present these difficulties of diagnosis.

Case 1

A man aged 69 was admitted as an emergency case on December 2, 1948, with a provisional diagnosis of strangulated right inguinal hernia. When seen after admission he was complaining of severe abdominal pain, which had come on suddenly the previous afternoon. He complained of nausea, and had vomited.

On examination he had a temperature of 100° F. (37.8° C.) and a pulse rate of 100. Examination of his abdomen revealed a tender irreducible swelling in the right inguinal region. His abdomen presented generalized rigidity, and bowel sounds were absent on auscultation. A provisional diagnosis was made of incarcerated right inguinal hernia, with probably strangulation and peritonitis.

Laparotomy (B. C. R.) revealed diffuse retroperitoneal extravasation of blood involving both the posterior and the anterior abdominal parietes. A large non-pulsatile mass was felt in the position of the abdominal aorta. It was then realized that the case was one of ruptured abdominal aneurysm, the external swelling being due to extravasated blood escaping through the inguinal canal.

After operation the patient's general condition deteriorated, and he died suddenly in the early hours of the morning of December 4.

Necropsy.—Heart:—The anterior descending branch of the left coronary artery showed extensive atheroma with ulceration and calcification. There was evidence of an old infarction at the apex of the heart. *Abdomen*:—The retroperitoneal tissues of the posterior abdomen showed a massive haemorrhage extending into the root of the mesentery. The haemorrhage was most pronounced on the right side, and in all there appeared to be approximately 3 pints (1.7 litres) of soft clot. The aorta showed extensive atheroma with ulceration and calcification, and in its lower part there was a large aneurysmal sac extending forwards. The sac measured approximately 10 cm. from above down, 6 cm. laterally, and 8 cm. antero-posteriorly. Its lining appeared to be continuous with that of the aorta, with superimposed masses of dense fibrin showing considerable calcification. Rupture had occurred on the right side of the sac, accounting for the greater haemorrhage in the retroperitoneal tissues on that side. The blood had tracked down along the iliac vessels on the right side through the femoral canal and along the spermatic cord. The aneurysmal bulge was entirely to the front of the aorta, and there was no evidence of any erosion of the vertebral bodies. It did not appear to be a dissecting aneurysm.

Case 2

On April 7, 1952, a man aged 76 was admitted to hospital as a surgical emergency with a diagnosis of strangulated left inguinal hernia. He gave a history of a painful lump in the left groin which had appeared for the first time only twenty-four hours previously. At the same time as the lump appeared he had some pain in the left iliac fossa. There was no nausea or vomiting. He was known to be hypertensive and subject to encephalopathic attacks.

On examination he was pale. His temperature was 98° F. (36.7° C.), pulse 114, and respirations 20. The abdomen was obese and moved well on respiration. No mass was felt, but there was deep tenderness in the left iliac fossa. Bowel sounds were normal. A tense, tender, irreducible swelling was presenting at the left external inguinal ring. A diagnosis of strangulated left inguinal hernia was made.

At operation (J. W. B.) the swelling was found to be an extravasation of blood into the layers of the cord, and on following this up a massive retroperitoneal haematoma was apparent. The patient made a good recovery from operation. While he was sitting up having his tea the following day he suddenly collapsed, and died within a few minutes.

Necropsy.—Post-mortem examination showed hypertrophy of the left ventricle and some atherosclerosis of both coronary arteries. The thoracic aorta showed scattered atheromatous ulcers and calcification of the wall, this condition extending into the abdominal part, where just above the bifurcation there was a large aneurysmal sac 6 by 4 by 4 in. (15 by 10 by 10 cm.). The wall of this had ruptured on the left side, causing a massive retroperitoneal haemorrhage extending from the diaphragm to the pelvis.

Pathology of Abdominal Aneurysm

Of aneurysms of the aorta those occurring in the abdominal part form only a small proportion. Thus Bryant (1903), searching the post-mortem records of Guy's Hospital covering 46 years, found that only 54 of 325 cases of aneurysm of the aorta had occurred in the abdomen. In reporting four cases of abdominal aortic aneurysm Jennings (1941) quotes Kampmeier (1936) as finding 386 cases reported in the literature.

The chief aetiological factors in the formation of abdominal aneurysms are syphilis, arteriosclerosis, and trauma. Syphilis is the most common cause in all the large series of aortic aneurysms reported, but a smaller series of cases in the older age group is associated with arteriosclerosis. Males predominate, and heavy manual work is a precipitating factor. The upper part of the abdominal aorta is the site of preference for the syphilitic cases, which are usually saccular. The arteriosclerotic aneurysms occur just above the bifurcation and may be fusiform or saccular in type.

The factors which predispose to aneurysm formation are a weakening of the arterial wall together with an increased pressure inside the lumen. Such increased pressure may be intermittent, such as occurs in sudden violent exercise, or it may be due to a persistent hypertension. The saccular aneurysm causes an out-pouching of the vessel at one point, whilst in the dissecting type the blood penetrates between the vessel coats and separates them.

Matas enumerates the terminal complications of abdominal aortic aneurysm as follows: (1) rupture of the aneurysmal sac; (2) disturbance of peripheral circulation, leading to gangrene of extremities; (3) fatal involvement of intestinal or renal circulation; (4) multiple small extravasations, leading to death from anaemia; (5) infection of the sac. with secondary haemorrhage; and (6) rupture into the inferior vena cava, with formation of an arteriovenous aneurysm and death from sudden arterial depletion.

However, whether of dissecting or saccular type, the aneurysm most commonly ends fatally with rupture. In the former type the rupture occurs suddenly, when the outer coat of the aorta is penetrated. The rupture of the saccular type occurs gradually, multiple small haemorrhages perhaps preceding the fatal massive rupture by several months.

Symptoms

Pain.—A dissecting aneurysm of the abdominal aorta may manifest itself suddenly with severe abdominal pain. The pain is generalized, but if the dissection extends into the iliac or femoral vessels it is commonly referred to both the lower limbs. If the dissection involves the lumbar arteries the pain will radiate to the loins. Similarly, extension of the dissection upward may cause radiation of the pain to the region of the chest and neck. After intraperitoneal rupture of a saccular aneurysm death ensues rapidly, and pain is not a prominent symptom. If, however, it should rupture extraperitoneally, as in the two cases reported, the pain may simulate that of a dissecting aneurysm in severity and distribution.

Haematemesis and Melaena.—This may occur in the dissecting type of aneurysm if the gastric or mesenteric vessels are involved. In the saccular type, involvement of and rupture into the intestinal tract may similarly produce the same symptoms. A case was recently reported by Barrett (1949).

Uraemia.—This may occur if the renal vessels are involved either by dissection or by propagation of intravascular thrombosis.

Signs

The clotting of small repeated extravasations, and intravascular clotting within the sac of the saccular aneurysm, may produce a mass which is palpable per abdomen. Intraperitoneal leaking may produce signs of peritonitis. In the case of a dissecting aneurysm involving iliac and femoral vessels there may be signs of bilateral circulatory insufficiency in the lower limbs. The downward extravasation in the retroperitoneal tissues of blood from a rupture of an aneurysm of either type may eventually reach the hernial orifices, as occurred in these two cases.

The only reference to similar cases in the literature is that reported by Eckert and Baker (1931), in which an extravasation into the right femoral canal led to the incorrect pre-operative diagnosis of psoas abscess.

Diagnosis

The only incontrovertible physical sign of an abdominal aortic aneurysm is a pulsatile swelling. In the presence of such protean manifestations as those mentioned, diagnosis can seldom be unequivocal.

The acute pain of the dissecting aneurysm and the leaking saccular aneurysm is the pain most likely to be misdiagnosed as one of the more usual types of acute abdominal emergency. The intense abdominal pain may mimic that of acute pancreatitis, perforated peptic ulcer, or mesenteric vascular occlusion. In such a case radiation of the pain into the loins, neck, or lower extremities may be significant. Previous symptoms of cardiovascular disease are usually absent, although the presence of hypertension may be suggestive.

The problem of the case which presents with extravasation at the hernial orifices is well illustrated in our two cases.

The concomitant occurrence of severe abdominal pain, lower-limb pain, and the presence of lower-limb vascular insufficiency may indicate a dissecting aneurysm, although similar signs may occur in a case of abdominal embolism.

Summary

Two cases of ruptured abdominal aortic aneurysm are recorded which presented clinically as incarcerated herniae.

The pathological and clinical features of abdominal aortic aneurysms are discussed.

The diverse modes in which these aneurysms, both ruptured and unruptured, may present are stressed and the differential diagnosis is indicated.

Our thanks are due to Mr. Clifford Jones for permission to publish Case 2, to Mr. Richard Charles for permission to publish Case 1 and to Mr. Trevor Shaw for a record of the necropsy findings.

REFERENCES

Barrett, G. M. (1949). *British Medical Journal*, **2**, 319.
Bryant, J. H. (1903). *Clin. J.*, **23**, 71.
Eckert, G. A., and Baker, R. E. (1931). *Nav. med. Bull., Wash.*, **29**, 667.
Jennings, G. H. (1941). *Lancet*, **1**, 719.
Kampmeier, R. H. (1936). *Amer. J. med. Sci.*, **192**, 97.

FOOD SUPPLY, BODY WEIGHT, AND ACTIVITY IN GREAT BRITAIN, 1943-9

BY

J. M. HARRIES, B.A.

Statistics and Intelligence Division, Ministry of Food

AND

DOROTHY F. HOLLINGSWORTH, B.Sc., M.I.Biol.

Scientific Adviser's Division, Ministry of Food

Between 1943 and 1949 a survey of the body weight of the population of Great Britain was made for the Ministry of Food with the object of providing an independent check on the adequacy of the national food supply. On certain broad assumptions a fall in body weight would have been a warning that food supplies were inadequate. Only gross changes in weight were being looked for, and none were found. There were, however, certain well-defined trends, and the opportunity has therefore been taken to determine whether these might themselves be related to trends in the energy value of the food consumed. The validity of such a comparison is bound to be influenced by changes in bodily activity such as those that might be associated with increased industrial productivity. As is shown below, attempts were made to arrive at an assessment of such activity, but without success. Nevertheless, the results recorded are so striking that it was felt that they warranted publication.

Experimental

The method of sample selection and the initial measurements of the Ministry's Body Weight Survey have already been described by Kemsley (1950, 1952), and a further paper describing the changes which occurred during the course of the survey is in the press. The survey was originally designed to cover the principal sections of the civil population over school age; as it continued, certain groups were omitted. The data used in this note refer to the two groups for which continuous records are available—industrial workers (male and female) in small firms and housewives. The former group was fairly well spread over the light industries, including some wholesaling and retailing trades. The latter group comprised working-class women who spent most of their time looking after their homes. For the present purpose only adults between 25 and 54 years have been included, because at ages outside these limits it is likely that growth or senescence will directly affect weight. Those individuals included in successive quarterly samples were compared, the weight changes thus obtained being averaged. These average changes, based on between 3,000 and 4,500 individuals, have been added cumulatively from the beginning of the survey, taking April, 1943, as the zero base-line. This quarterly index is shown by the dotted line in Figs. 1 and 2.

There are several sets of data referring to food consumption which might be compared with these graphs. Total supplies of foods as published in the various reports on Food Consumption Levels (Ministry of Food, 1949, 1951a) are not suitable, because they are complicated by changes in retail and household stocks and therefore do not adequately reflect current consumption. Changes in the ration scales are not realistic because they do not include unrationed foods or foods which were sold against points. For the present purpose, therefore, data from the Ministry of Food's National Food Survey have been used. These recorded the actual domestic food consumption of urban working-class households. The relevant results, together with details of the method of sampling and the techniques used, have already been published (Ministry of Food, 1951b). The data were based on actual purchases of foods and changes in larder stocks, and from these the average energy values of the food consumed could be calculated for comparison with the results of the body weight survey. No allowances have been made for food eaten in catering establishments, or for certain items, such as sweets, ice-cream, alcoholic and some soft drinks, for which the housewife could not readily provide complete data. Nor, on the other hand, are allowances made for food eaten in the home by visitors. These omissions do not, however, invalidate the figures for the present purpose, since on the average their energy values did not vary appreciably during the years under review.

Fig. 3 shows the resulting estimated changes in food consumption between 1943 and 1949, based on some 20,000 to 30,000 individuals per year. The dotted line shows changes in quarterly averages of the energy value of the food available for consumption in the home per head per day. It has been constructed on the same basis as the graphs in Figs. 1 and 2, changes being recorded from an arbitrary zero base-line starting at April, 1943.

In all three figures the medium-term trend line has also been calculated, based on a centralized moving average of four successive readings. The purpose of this, shown as a thicker continuous line on the graphs, has been to eliminate seasonal fluctuations.* Both body weight and calories varied seasonally in a fairly regular manner, but such variations were regarded as extraneous to the present purpose, which is to consider medium-term trends.

The simpler statistical methods of correlation are not applicable to time series without modification, and it is not

*It is known that under normal circumstances seasonal, and roughly parallel, fluctuations occur in body weight and food consumption. It was important, therefore, to eliminate these in considering longer-term trends.

possible to use the special methods devised for the correlation of time series to measure the agreement between the medium-term trends of these graphs, mainly because the series are not long enough—that is, there are too few readings. It would have been feasible to calculate a correlation co-efficient for the seasonal fluctuations, but, for the reason stated above, this would have been irrelevant. As both body weights and consumption levels are derived from samples of typical urban working-class groups their results may be validly compared.

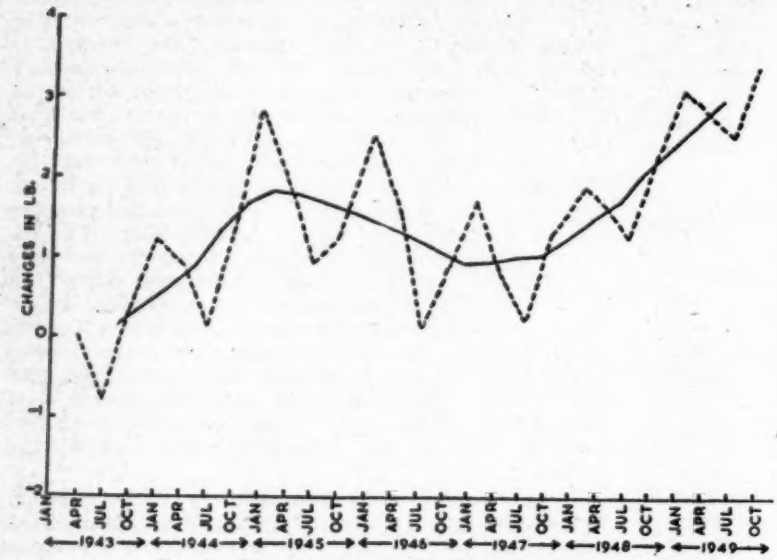

CHANGES IN LB.
4
3
2
1
0
-1
-2
JAN APR JUL OCT JAN APR JUL OCT JAN APR JUL OCT JAN APR JUL OCT JAN APR JUL OCT JAN APR JUL OCT
1943 1944 1945 1946 1947 1948 1949

FIG. 1.—Changes in weight of adult males, 1943–9.

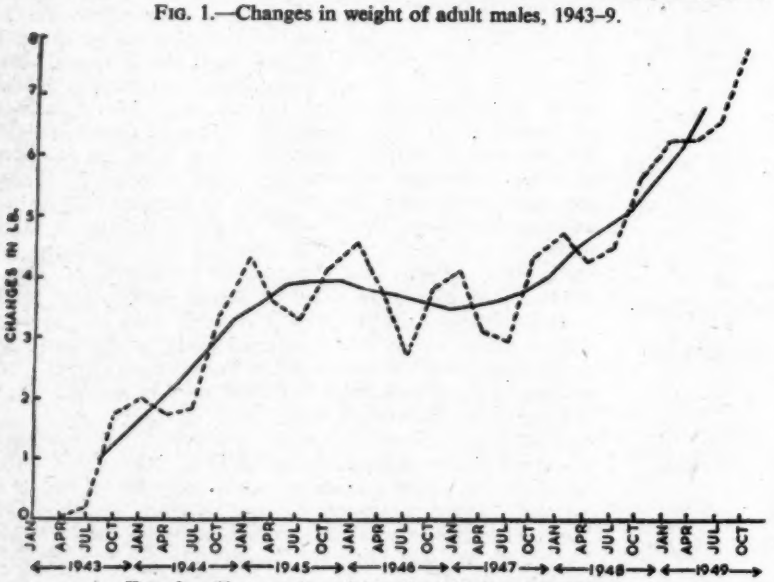


FIG. 2.—Changes in weight of adult females, 1943–9.

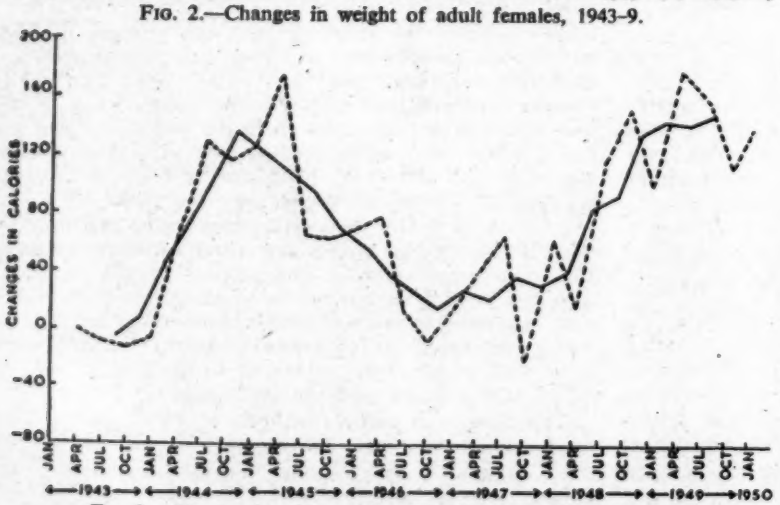

CHANGES IN CALORIES
200
160
120
80
40
0
-40
-80
JAN APR JUL OCT JAN APR JUL OCT JAN APR JUL OCT JAN APR JUL OCT JAN APR JUL OCT JAN APR JUL OCT JAN
1943 1944 1945 1946 1947 1948 1949 1950

FIG. 3.—Changes in energy value of working-class diets, 1943–9.

Results

Visual comparison shows a very striking resemblance between the graphs. In each there are three fairly clear phases. The first extends from April, 1943, to the beginning of 1945; the second from the beginning of 1945 to the beginning of 1947; and the third from the beginning of 1947 to the end of 1949. It will be seen that in all three phases the changes in body weight closely parallel the changes in the energy value of the foods consumed. Thus in the first phase the energy value increased by about 140 calories† per head per day, while the body weight rose by about 1½ lb. (680 g.) for men and 3 lb. (1,360 g.) for women. During the second phase, when there was a decrease in the number of calories per head per day of about 120, the body weight fell by about 1 lb. (454 g.) for men and ½ lb. (227 g.) for women, the latter starting to lose weight slightly later than the men. During the third phase, when the energy value of the food consumed again increased by about 120 calories, the body weight again rose by 2 lb. (907 g.) for men and 3 lb. (1,360 g.) for women. It will be noted that the general trends of the graphs in Figs. 1 and 2 are practically parallel during the first and third phases. The fall in weight during the second phase is statistically significant for both men and women.

Although there have been some inquiries into the body weights of adults under peace-time conditions of relative abundance, none of them has examined *changes* in weight, so that no data are available to determine whether the increases in weight during the first and third phases are normal for adults. As each was preceded by a period of stringency in the food supply—the calorie values of total food supplies for 1940–3, like those for 1946–7, being below 2,900 per head per day (Ministry of Food, 1949)—it is possible that each was rather greater than normal, a conclusion which is supported by Kemsley's (1950) comparison with earlier data.

It has been stated that Figs. 1 and 2 are based on two groups—industrial workers (male and female) in small firms and housewives. The former group were drawn from firms with and without canteens, and it was therefore possible to examine their weight increases separately (Kemsley, 1950). It was found that the greatest fall in weight took place amongst housewives and those workers who had no access to canteen facilities. The remainder, who had access to canteens, showed either a small loss in weight only, or even a small gain.

Growth Rates of Children

It is well known that unexplained secular changes, apparently unconnected with food supplies, occur in the growth rates,

†This and subsequent calorie values given in this paper are calculated according to the convention adopted by the Medical Research Council (1945).

and hence the body weights, of children. Nevertheless it seemed desirable, in considering the decline in body weight of adults in 1945 and 1946, to determine whether there had been any parallel changes in the growth rates of children. Changes in growth rates have varied in different parts of the country; Liverpool and London are examples noted by the British Medical Association's Committee on Nutrition (1950). The chief sources of information are the annual reports of school medical officers and special inquiries by the Ministries of Education and Health which have been summarized by Bransby (1944, 1946). From these and from the later reports quoted below the trends of changes in the growth rate of schoolchildren may be summarized as follows. Growth rates were well maintained during the war years among children living at home. There were small decreases in the growth rate (fractions of a pound and of an inch) in some localities in 1940 and 1941, and even cases of actual loss in weight as compared with pre-war, chiefly amongst older children, but these had been rectified by 1943. The groups showing the greatest fall made the quickest recovery, and the improvement was continued up to the end of 1945. There is evidence that this improvement was not maintained in 1946 and 1947.

Examples are to be found in the Report on the Medical Inspection and Treatment of School Children for the year ended July 31, 1950, for the Corporation of Glasgow (1950), which shows (p. 93) that the weights of 13-year-olds in 1946 were slightly less than those in 1945, and that the weights of 5-year-olds in 1947 were also less than those in the preceding year; in the Report on the work of the School Health Service for the year 1950 for the City of Liverpool Education Committee (Frazer, 1950), where graphs (p. 15) for comparative weights of boys and girls aged 5, 8, and 12 show greater but similar changes in weight in 1946 and 1947 (the shapes of these graphs are similar to those of Figs. 1 and 2); and in the Report of the School Medical Officer of the London County Council (Daley, 1948), where some reductions in weight in 1947 are recorded. Although these inquiries did not all demonstrate statistically significant decreases in growth rate during 1946 and 1947, the fact that the trends are similar in each confirms the view that there was some retardation. The figures for 1948 onwards show that in all the areas quoted above a definite recovery in the growth rate had set in. It is instructive to compare these fluctuations in growth rate with similar and, in some instances, more pronounced changes which have been reported from several European countries at the time of severe food rationing—for example, Trémolières and Boulenger (1950); *Tijdschrift voor sociale geneeskunde* (1945).

Discussion

As mentioned in the opening paragraph of this paper, the validity of any comparison between food consumption and changes in body weight is apt to be influenced by changes in bodily activity such as those which might be associated with increased industrial productivity. Thus Kraut and Müller (1946) showed that a change in the calorie value of the food supply affected both body weight and productivity, and that when the calorie value was held constant but non-food incentives were offered, body weight and productivity varied inversely.

In Kraut and Müller's experiments the workers were, however, engaged under strict supervision in shovelling waste matter from trucks on to an embankment—a form of work in which the measurement of productivity can be taken as a direct index of bodily activity. On a national scale this simple relationship no longer holds, since industrial productivity is influenced by many factors other than the degree of muscular work—for example, by changes in the efficiency of management, by the development of labour-saving devices, and, on the broader scale, the availability of raw materials and the redistribution of man-power among different industries. Moreover, such indices of industrial productivity as are available could not in any event be used to assess total bodily activity. For instance, figures per man-hour or per man-shift make no allowance for extra hours worked or for absenteeism, both of which would affect bodily activity on a national scale. Again, the classifications used as a basis for assessing total production and employment between different industries have been recorded on different bases during the period covered by the present study (the Standard Industrial Classification was not introduced until 1949), so that no continuing index is available.

Such evidence as there is for the years 1943–8 suggests that in any event changes in the nation's industrial productivity were relatively small (Rostas, 1948). It is, moreover, relevant to point out that the present study included not only workers in the light industries but also housewives, whose activities are very unlikely to have varied appreciably over the period covered. It is therefore not unreasonable to assume that bodily activity may be regarded as having remained constant and that a direct comparison of food consumption and body weight is valid. It is true that any violent change in food consumption might itself affect the energy output of the body. It seems clear, however, that the changes in diet which actually took place would have been quite insufficient to affect either the specific dynamic action of the foods eaten* or the average basal metabolic rates of individuals within the population. Nor is there reason to believe that the body weights, which are dependent on water content as well as on food consumption and muscular activity, could have been appreciably affected by changes in water balance.

If these assumptions are accepted the body weight data presented here can justifiably be used as at least an approximate index of the adequacy of the diet. In an earlier publication (Special Joint Committee of the Combined Food Board, 1944) a suggestion, based on evidence of the early years of the war, was made that a food supply providing 2,800 calories per head per day "is for the United Kingdom about marginal." Comparison of the relation between the food-consumption levels and the body weight changes recorded in this paper and the calorie value of total supplies of food moving into civilian consumption (Ministry of Food, 1949, 1951a) shows that during 1944, when the calorie value of the total food supply was just over 3,000 per head per day, adult men and women gained weight; that during 1945, when the calorie level was over 2,900, weight was roughly constant; that during 1946 and the early part of 1947, when the calorie level fell below 2,900 and dissatisfaction over the food supply was voiced publicly, adults lost weight. In 1948, when the calorie level had again risen above 2,900, the trend of 1946 and 1947 was reversed. As already noted here, the trends for the growth rates of children were very similar. These comparisons suggest that, in order to maintain full working efficiency and morale, the apparently normal slight annual increase in weight in adults, and the satisfactory growth of children, the marginal figure of 2,800 put forward in 1944 is probably too low. When the supply was just under 2,900 the differential weight loss as between those with and without canteen facilities reflects the marginal nature even of a supply of 2,900.

Because it is notable that the decreases in body weight described here were not accompanied by any measurable ill effects to health (Magee, 1946; British Medical Association, 1950) it seems that body weight can be used as an earlier index of inadequacy in food supplies than clinical examinations. It is true that temporary weight losses such as those recorded in 1945 and 1946 would probably have been too slight to have had any direct detrimental effect on health. But it is reasonable to assume that if such weight losses were to persist for longer periods they could not be safely neglected, not only from the health aspect but from the point of view of the morale of the population and of their potential effects on productivity.

*The range in the daily supply of protein (the greatest factor in determining specific dynamic action of the total food consumed) between 1943 and 1949 was only 5 g. per head (Ministry of Food, 1949, 1951a, 1951b).

Summary

The average changes in weight of groups of industrial workers (male and female) in small firms and of housewives measured during the course of the Ministry of Food's Body Weight Survey (1943 to 1949) have been compared with the calorie value of the food consumption recorded for urban working-class households in the Ministry's National Food Survey during the same years. Body weights of adults fluctuated with the calorie values of the diet. There is evidence from various parts of the country of parallel changes in the growth rates of children.

The bearing of these results on the desirable calorie levels of the national food supply is discussed.

REFERENCES

Bransby, E. R. (1944). *Monthly Bull. Minist. Hlth, Lond.*, 3, 212.
—— (1946). Ibid., 5, 57.
British Medical Association (1950). Report of the Committee on Nutrition. London.
Corporation of Glasgow (1950). Report on the Medical Inspection and Treatment of School Children for the Year ended July 31, 1950. Health and Welfare Department.
Daley, Allen (1948). *Med. Offr*, 79, 242.
Frazer, W. M. (1950). Report on the work on the School Health Service for the year 1950. Education Committee, City of Liverpool.
Kemsley, W. F. F. (1950). *Ann. Eugen., Camb.*, 15, 161.
—— (1952). Ibid., 16, 316.
Kraut, H. A., and Müller, E. A. (1946). *Science*, 104, 495.
Magee, H. E. (1946). *British Medical Journal*, 1, 475.
Medical Research Council (1945). Nutritive Values of Wartime Foods. War Memorandum No. 14. H.M.S.O., London.
Ministry of Food (1949). Food Consumption Levels in the United Kingdom (Cmd. 7842). H.M.S.O., London.
—— (1951a). *Bull. Minist. Food*, No. 611, p. 4.
—— (1951b). First report of the National Food Survey Committee. The Urban Working Class Diet, 1940 to 1949. H.M.S.O., London.
Rostas, L. (1948). *The Times*, November 10-11. London.
Special Joint Committee, Combined Food Board (1944). Food Consumption Levels in the United States, Canada, and the United Kingdom. H.M.S.O., London.
Tijdschrift voor sociale geneeskunde (1945). Nos. 1 and 4 Onderzoek naar den Voedingstoestand van Schoolkinderen te Amsterdam, 1935-45.
Trémolières, J., and Boulenger, J. J. (1950). *Rec. Inst. nat. Hyg., Paris*, 4, 117.

HAEMOLYTIC ANAEMIA TREATED BY A.C.T.H., CORTISONE, AND SPLENECTOMY

BY

J. D. AITCHISON, M.B., F.R.F.P.S., M.R.C.P.
Senior Medical Registrar, Aberdeen General Hospitals

Encouraging results have been reported in the treatment of acquired haemolytic anaemia with adrenocorticotrophic hormone (A.C.T.H.), cortisone, and compound F (Gardner, 1950; Dameshek *et al.*, 1951; Davidson *et al.*, 1951; Rosenthal *et al.*, 1952). It has been suggested that this type of therapy succeeds in cases in which an abnormal antibody can be demonstrated (Dameshek, 1950), but the exact mechanism of action is still quite obscure.

A case of this type, encountered recently, has been treated with A.C.T.H., with cortisone, and, finally, by splenectomy. In reviewing the management of this patient several points of practical importance have emerged; they are reported here in the hope that this experience may be of value in the future conduct of such cases.

Case Report

A schoolteacher aged 63 was admitted to hospital on May 1, 1951, with severe anaemia. There was no family history of jaundice or anaemia. In 1942 he became jaundiced because of gall-stones, and cholecystectomy was performed. The nature of the stones is not known. A year later, when in hospital with a spontaneous pneumothorax, his haemoglobin was 82% and red cells 5,000,000 per c.mm. In 1947 he became anaemic, and on June 19 of that year his haemoglobin was 40%, red cells 1,500,000 per c.mm., and colour index 1.3. A blood film showed "megalocytosis," and pernicious anaemia was diagnosed. He was given liver injections, and felt well until early in 1951, when he again became anaemic, despite treatment with liver and vitamin B_{12}.

On admission he was pale and slightly jaundiced. The liver edge was palpable, and the tip of the spleen could be felt. Blood examination showed: haemoglobin, 34%: red cells, 1,090,000 per c.mm.; colour index, 1.6; reticulocytes, 16%; white cells, 10,300 per c.mm. The differential white-cell count was within normal limits. There was macrocytosis and polychromasia of the red cells. Examination of the sternal marrow showed normoblastic hyperplasia.

Autoagglutination of the blood was noted. A direct Coombs test was positive. Autohaemagglutinins were present in a titre of 64 at refrigerator temperature, 8 at room temperature, and 2 at body temperature. The patient's blood was Group O Rh-positive. Red-cell fragility was within normal limits. Serum bilirubin was 2.2 mg. per 100 ml. There was a positive indirect van den Bergh reaction. The urine contained a trace of albumin and increased urobilinogen. Stool benzidine tests were negative. A fractional test meal showed a histamine-fast achlorhydria. Thymol turbidity was 2.5 units and plasma proteins 7.6 g.% (albumin/globulin ratio 1.25:1). Serum alkaline phosphatase was 75 units (King–Armstrong), and E.S.R. (Westergren) 150 mm. in the first hour.

On May 8 the haemoglobin was 32%. On the 10th a transfusion of Group O blood was started, but was discontinued because of a severe febrile reaction after a third of a pint (190 ml.) of blood had been given. On the 12th the patient was given 25 mg. of A.C.T.H. intramuscularly, six-hourly, and this was continued for three days. There was no apparent effect on the disease (Fig. 1) or on the eosinophil count even when the dose was increased to 40 mg. six-hourly. Three pints (1,700 ml.) of blood was transfused uneventfully on May 18. The haemoglobin level continued to fall, and on May 25 a further five pints (2,740 ml.) of blood was given. The patient was so ill that a proper interval for the observation of the effects of the transfusion could not be allowed, and 300 mg. of cortisone was given on May 27, 200 mg. on the next two days, and 100 mg. on the following four days. Signs of haemolysis diminished. The E.S.R. fell, autoagglutination of the blood became much less evident, and the haemoglobin level rose (Fig. 1). The improvement continued until the dose of cortisone was reduced to 25 mg. daily, when the E.S.R. rose and signs of haemolysis increased.

A second course of cortisone was started on June 19, and this also produced improvement, but less than had followed the first course. Again, as the dose of cortisone was reduced to 25 mg. daily, the E.S.R. rose, autoagglutination of the blood became more evident, signs of haemolysis increased, and the haemoglobin level fell. The dose of cortisone was increased a third time to 300 mg. daily, with even less effect, and, although the daily intake was maintained above 50 mg. (Fig. 2), the haemoglobin fell steadily until splenectomy was performed by Mr. S. G. Davidson on July 27. On the two days following operation autoagglutination was virtually absent, but reappeared thereafter. Signs of haemolysis diminished and the haemoglobin rose steadily. On August 8 the wound burst, and there was prolonged delay in its healing. Cortisone was discontinued on August 28, and the haemoglobin fell slightly after this. The E.S.R. rose again. Autoagglutination of the blood

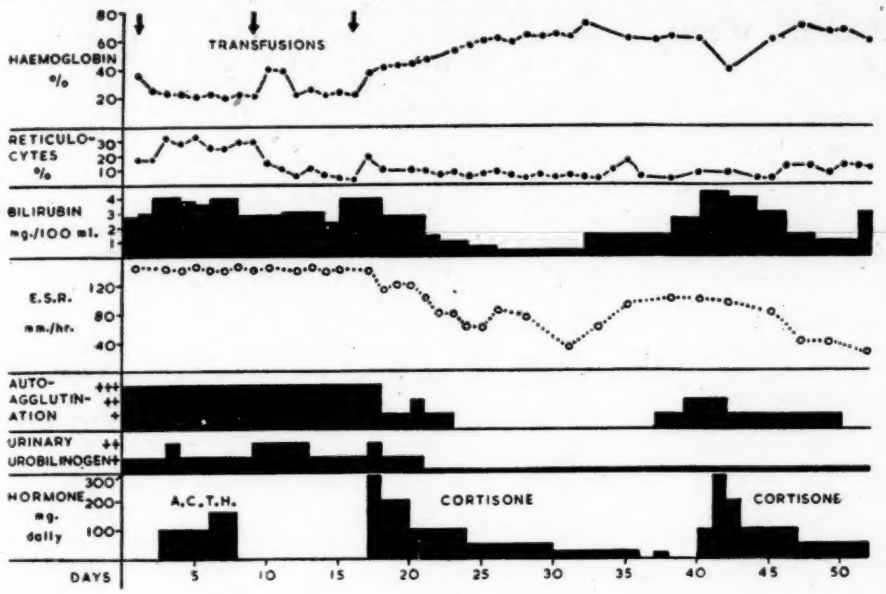


FIG. 1

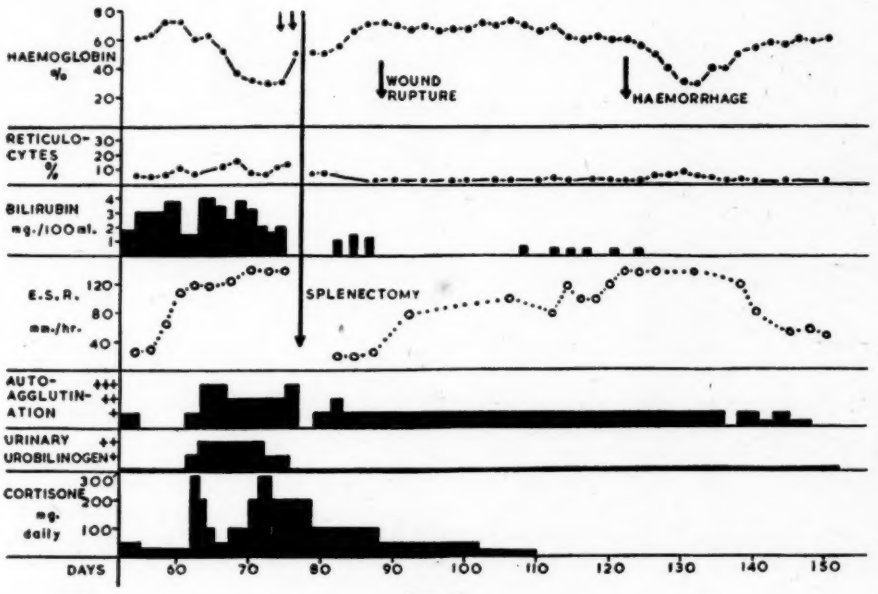


FIG. 2

was still evident. On September 8 there was an arterial haemorrhage from the operation wound and the patient was critically ill for several days. A week later the haemoglobin began to rise again and his progress was uneventful from that time. In early November his haemoglobin was 80%, his wound had healed, and he was well enough to walk a short distance. The E.S.R. was then 80 mm. in one hour. He has continued well up to the time of writing. Autoagglutination of the blood was still present, but was slight in degree. The E.S.R. has gradually decreased to 18 mm. in one hour.

Comment

The failure of A.C.T.H. to influence the haemolytic process in this case is of some interest. Despite the relatively large dose administered (160 mg. daily), there was no adrenal response, as judged by eosinophil counts. It must be presumed that this patient's adrenal glands were incapable of responding to even this degree of stimulation, since 25 mg. of the same batch of A.C.T.H. subsequently gave a positive Thorn test in another patient.

Cortisone initially produced dramatic improvement, reducing haemolysis and autoagglutination, and causing a fall in the sedimentation rate. It is of great interest, however, to note that subsequent courses of the hormone had progressively less effect. In retrospect, it would clearly have been wiser to have taken advantage of the first remission produced by cortisone to remove the spleen, since the operation had eventually to be performed as a matter of desperate expediency. It should also be pointed out that this patient's operation wound was manifestly slow in healing, and the bursting of the wound came near to being a fatal disaster. These complications may well have been due to the prolonged administration of cortisone. Howes *et al.* (1950) and Bangham (1951) have shown experimentally that cortisone causes delay in healing and readiness to rupture in surgical wounds. Thus, although the action of cortisone was initially life-saving, the continuation of the drug may have added materially to the danger of splenectomy when the operation was eventually undertaken.

Summary

A case of idiopathic acquired anaemia treated with A.C.T.H., cortisone, and splenectomy is described.

I wish to thank Dr. R. J. Duthie for permission to publish this case. I am indebted to Dr. H. N. Robson for much helpful advice in the management and presentation.

REFERENCES

Bangham, A. D. (1951). *Brit. J. exp. Path.*, 32, 77.
Dameshek, W. (1950). *Blood*, 5, 779.
—— Rosenthal, M. C., and Schwartz, L. I. (1951). *New Engl. J. Med.* 244, 117.
Davidson, L. S. P., Duthie, J. J. R., Girdwood, R. H., and Sinclair, R. J. G. (1951). *British Medical Journal*, 1, 657.
Gardner, F. (1950). *Blood*, 5, 791.
Howes, E. L., Plotz, C. M., Blunt, J. W., and Ragan, C. (1950). *Surgery*, 28, 177.
Rosenthal, M. C., Spaet, T. H., Goldenberg, H., and Dameshek, W. (1952) *Lancet*, 1, 1135.

The International Diabetes Federation recently published the report of its first congress, held at Leyden on July 7–12, 1952. The report includes the inaugural address by Professor C. H. Best (honorary president of the Federation) and abstracts of some 33 papers read at the congress with indications, when possible, where they have been or will be published *in extenso*. Matters discussed by the council of the Federation included proposals for a study of the geographical distribution of diabetes, individual social help for diabetics, hostels for diabetic children, insurance against risks of diabetic coma, and driving licences for diabetics.

DIFFUSE PHLEBOSCLEROSIS WITH CALCIFICATION

BY

A. CHARAN, M.S.

Lecturer in Surgery, K.G. Medical College, Lucknow

An unusual case of diffuse phlebosclerosis with calcification is presented. No similar case has been found in the literature.

Case History

A Hindu male aged 20 was admitted on March 9, 1951, with increased frequency of micturition, dysuria, and haematuria of two days' duration. He also complained of lumps in front of the elbows and behind the knees, which had apparently first been noticed by his parents soon after his birth and had since been slowly increasing in size. He had had dysentery during childhood and two recent attacks of asthma. He had also had two severe attacks of renal colic,

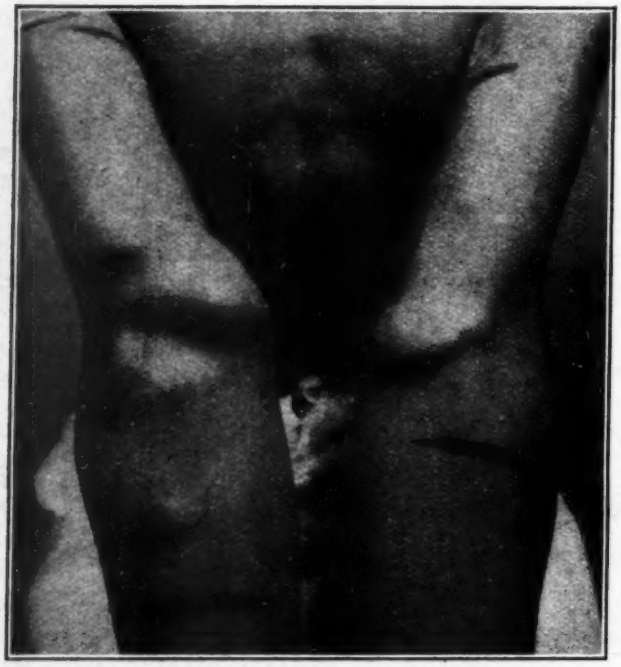


FIG. 1.—Photograph showing both arms and forearms with bony-hard cord-like swellings. Their extent is marked.

four years and two and a half years previously, lasting several hours each; haematuria had not been present on these occasions. There was no relevant family history.

On examination the patient was shocked and restless with a pulse rate of 120. There were bilateral, bony-hard, nodular, cord-like swellings in the lower two-thirds of the arms, extending across the cubital fossa into the anterior aspect of the upper half of the forearm (Fig. 1). The swellings were fixed to the deeper structures, and related to the main vascular bundle of the limb. The brachial and radial pulses were very feeble on both sides, but the arterial walls were not palpably thickened. The axillary pulse was normal. Clinically the swellings appeared to be calcified venous varices. Similar swellings were present bilaterally in the legs, extending from the lower third of the thigh, across the popliteal fossa, to half-way down the back of the calf. The pulsation of the popliteal and posterior tibial arteries was greatly reduced on both sides, but that of the femoral artery was normal. There were no signs of peripheral vascular insufficiency. No other physical abnormalities were discovered except a urethral stricture 1½ in. (3.8 cm.) proximal to the urinary meatus.

Investigations.—Urine, S.G. 1020; albumin, +; large numbers of red blood cells; sterile on two occasions. Renal function: non-protein nitrogen, 50 mg. per 100 ml.; blood urea, 43 mg. per 100 ml.; urea concentration, 2.2%. Blood: white cells, 15,650 per c.mm. (poly. 83%, lymph. 9%, eosin. 2%, mono. 6%); red cells, 3,380,000 per c.mm.; Hb, 54% (8.1 g.); slight anisocytosis and moderate vacuolation also present; bleeding-time, 2 minutes 55 seconds; coagulation time, 5 minutes 15 seconds (both within normal limits); serum calcium, 10.2 mg. per 100 ml.; serum phosphorus, 4 mg. per 100 ml.; alkaline phosphatase, 6 units (similar results on two occasions); Sulkowitch's test, no increase of urinary calcium excretion; Kahn test and Wassermann reaction negative.

Radiological examination.—Plain *x*-ray pictures of the arms (Fig. 2), legs (Fig. 3), head and neck, and renal pedicle

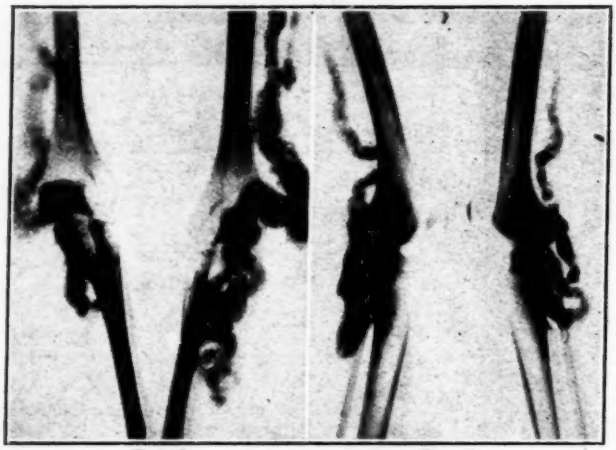


FIG. 2. FIG. 3.

FIG. 2.—Radiograph of arms, showing radio-opaque cord-like shadows of calcified veins. FIG. 3.—Radiograph of legs showing radio-opaque cord-like shadows of calcified veins.

showed calcified cord-like shadows, provisionally diagnosed as calcified veins. Descending pyelography showed both kidneys; the left pelvis and upper part of the ureter were not so well seen as on the right; the dye reached the bladder.

Treatment and Operative Investigations.—For the haematuria he was given three blood transfusions (totalling 1,050 ml.), bladder irrigations, and coagulants such as calcium, "coagulin," and vitamin K. The swelling in the left arm was excised (Fig. 4). It was a calcified tortuous mass, replacing the brachial vein and adherent to the brachial artery and median nerve. The brachial artery and the other venae comitantes were normal. The cut surface of the specimen was yellowish white, granular, and gritty to the touch. Histological examination showed a circular mass of fibrous tissue in which were several foci of calcium deposit, each surrounded by dense collagenous tissue. In the fibrous bands between the calcium deposits there were small capillaries. No elastic or muscular tissue was seen. The parathyroid glands were explored, but were macroscopically normal. Pyelolithotomy was undertaken on the right side, but, apart from the presence of a calculus and calcified renal veins, no other abnormality was discovered.

FIG. 4.—Specimen of the cord-like hard tortuous vein removed at operation from the left arm. The arrow shows the normal vena comitans.

Discussion

The clinical nature of the swellings, together with *x*-ray and histological

findings, leaves little doubt that they were venous varices, whose lumen had become obliterated and undergone secondary calcification.

The differential diagnosis comprised parathyroid dyscrasia with disturbed calcium metabolism; other causes of pathological calcification; and primary degenerative vascular conditions with secondary calcification. Parathyroid dyscrasia is ruled out by the negative clinical, biochemical, radiological, and surgical findings. Phlebolith, calcified parasites, myositis ossificans, etc., can be excluded on the physical character of the lumps, the *x*-ray findings, and the absence of their respective signs and symptoms. Of the degenerative vascular conditions, arteriosclerosis and Mönckeberg's degeneration can be excluded in view of the age of the patient and the absence of evidence of any organic changes in the arteries.

A diagnosis of phlebosclerosis is thus very appropriate, and most of the features of this case can be explained by it.

A review of the literature on phlebosclerosis shows that reports of the condition are uncommon. Before 1905 there is hardly any reference to it, and it had been described as occurring in elderly persons and associated with patchy subintimal fibrosis, calcification being unknown. Martin and Meakins (1905), Stahl and Zeh (1923), and Hauswirth and Eisenberg (1931) defined the condition more closely, calling it venofibrosis, peripheral phlebosclerosis, hyperplastic endophlebitis, etc. They describe it as of slow and gradual course and that it is seen in patients between 18 and 45 years of age. It has been associated with cases of gout, lead-poisoning, and syphilis. Hauswirth and Eisenberg found a high incidence of the condition in peptic ulcer cases, but did not encounter oedema, necrosis, or calcification. Histologically they found hyalinization of the intima and hyperplasia of fibrous tissue in the media, leading to obliteration of the lumen. It was progressive and painless. Boyd (1947) has described phlebosclerosis as occurring between the ages of 20 and 30, unassociated with arteriosclerosis, vascular insufficiency, or inflammation. Fatty degeneration or calcification is always bilateral, and affects both superficial and deep veins, which feel like hard mobile cords. The lumen is narrowed and there is medial fibrosis. Levin and Bucy (1936) have described a case of proliferative endophlebitis with phlebosclerosis: superficial veins were firm, small, cord-like, and not varicose.

In the present case the swellings were first noticed soon after birth, and there is therefore a strong suspicion of their being congenital. Their pathogenesis can be explained only on the basis of some abnormal developmental process. Possible hypotheses are: (1) that calcification has occurred in areas of abnormal vascular formation where canalization has not taken place; or (2) at a later stage, after vein formation, ischaemia has occurred because of an inadequate blood supply from the vasa vasorum, with subsequent calcification. The presence of renal calculus is difficult to explain, and may be coincidental.

I am indebted to Professor S. N. Mathur, head of the department of surgery and superintendent, Gandhi Memorial and Associated Hospitals, for permission to publish the case, and to Professor S. C. Misra, professor of clinical surgery, Professor V. S. Manglik, professor of pathology, and to other colleagues for their help and advice.

REFERENCES

Boyd, W. (1947). *Textbook of Pathology*. London.
Hauswirth L., and Eisenberg, A. A. (1931). *Arch. Path.*, 11, 857.
Levin, P. M., and Bucy, P. C. (1936). *Arch. intern. Med.*, 57, 787.
Martin, C F., and Meakins, J. C. (1905). *Amer. Med.*, 10, 611.
Stahl, R., and Zeh, F. (1923). *Virchows Arch. path. Anat.*, 242, 70.

The tendency for certain organizations to raise hopes of adoption before the birth of an illegitimate child is condemned in the recently issued annual report of the National Council for the Unmarried Mother and her Child. This sometimes leads to inadequate antenatal preparations on the part of the mother, since she feels certain that she will give up her child on leaving hospital. However, once the baby has arrived she often changes her mind.

Medical Memorandum

Neonatal Death due to Ligation of Umbilical Cord by an Amniotic Band

The following case of neonatal death is interesting enough to be placed on record. So far as we can ascertain no previous foetal death due to this exact cause has been published.

CASE REPORT

The patient was a healthy multipara aged 26, with one previous live baby. Her first confinement, four years previously, was conducted by another practitioner, and was normal in every respect.

Her second pregnancy was associated with much sickness in the early months. The presentation in the early part of the eighth month was a breech, which underwent spontaneous version. The final presentation, confirmed by *x*-ray examination, was a normal vertex. The expected date of delivery was about March 28, 1951. There was no clinical evidence of hydramnios, and the case was regarded as a normal pregnancy.

Foetal movements were vigorous up to the morning of March 17, when they suddenly stopped. Unfortunately an opportunity of examining the patient at this stage did not arise, and she went into labour in the early hours of March 18. Labour proceeded rapidly, and the passage of meconium was noted by the nurse when the membranes ruptured at 4.50 a.m. One of us was summoned by the nurse, and on arrival about 30 minutes later a dead baby had just been delivered. So far as we can judge, the first stage took about three hours and the second stage about three-quarters of an hour.

The appearance was very remarkable. The placenta had not yet been delivered, and issuing from the vagina was a thin cord-like structure which was knotted tightly round the umbilical cord about 5 in. (12.5 cm.) from the umbilicus. The baby, a male of 6 lb. 10 oz. (3 kg.) was not macerated and appeared normal. The nurse had cut the cord proximal and distal to the constricting band. The placenta was delivered after a normal period without any difficulty.

Examination of the placenta and the cord-like band showed that the latter arose from the amnion and that its length from the edge of the placenta to the point of constriction on the cord was 22 in. (56 cm.). Its total length was 31 in. (78.5 cm.). The appearance is well demonstrated in the accompanying photograph.

A possible explanation of the mechanism of the constriction is that the umbilical cord had been encircled by the amniotic band in a loose knot during spontaneous version, and that the constriction was fatally tightened with the descent of the head into the pelvis about or immediately before the onset of labour. This would seem to be borne out by the fact that the foetal movements suddenly ceased about 15 hours before the onset of labour.

F. O'K. CRAVEN, M.D.
C. J. M. GEDDES, M.B., Ch.B.

Reviews

OESTROGENS AND NEOPLASIA

Oestrogens and Neoplasia. By Harold Burrows, C.B.E., Ph.D., F.R.C.S., and Eric S. Horning, M.A., D.Sc. With contributions by W. C. J. Ross and C. W. Shoppee. (Pp. 189. £1 10s.) Oxford: Blackwell Scientific Publications. 1952.

Dr. Charles Huggins, in a foreword to the book, states: "The authors present many complex data which they skilfully integrate in a holistic approach which deserves the thanks of all workers in cancer and endocrinology," and the reviewer would find it difficult to improve upon this comment. The authors are well known in the field of experimental cancer research, and therefore we should be right in expecting a work of outstanding quality from them. They have very wisely brought in workers who are expert in some of the highly specialized fields. The first chapter, on the chemistry of the oestrogens, is by Professor C. W. Shoppee. In the reviewer's opinion it is a great pity that Professor Shoppee does not discuss the naturally occurring hormones at greater length, for his account of the naturally occurring oestrogens is so concentrated that only the expert would be able to understand it. It would seem that this work is bound to have a number of editions, and we hope that in the next one Professor Shoppee will give readers the benefit of his great ability and knowledge in explaining the complicated structure of these steroidal hormones. He discusses fully and with complete clarity the various synthetic oestrogenic substances, and gives a detailed account of the various researches leading to the production of stilboestrol. He concludes with a very good summary of the recent work on the metabolism of oestrogens.

Chapter 2 is on the source of oestrogens, and Chapter 3 is a general account of the biological action of these substances, particularly on tissue growth. Chapters 4 to 12 are on tumours in various parts of the body, and the relationship of oestrogens to them. Chapters 13 and 14 are concerned mainly with the hereditary factors in mammary cancer, and Chapter 15 with the therapeutic application of oestrogens in various forms. Chapter 16, by W. C. J. Ross, is concerned with the chemotherapy of cancer, and covers urethane, folic acid antagonists, and other substances. In the final chapter Dr. Burrows discusses tumours that secrete oestrogens; it contains a valuable and interesting collection of data. The volume closes with an excellent series of references.

E. C. DODDS.

ACTINOMYCOSIS IN GENERAL PRACTICE

What the General Practitioner ought to Know about Human Actinomycosis. By V. Zachary Cope, M.S., F.R.C.S. (Pp. 80; illustrated. 12s. 6d.) London: William Heinemann Medical Books. 1952.

Mr. Cope has set out to do exactly what he has promised—namely, to paint such a picture of the pathological result of infection with actinomycosis that the busy general practitioner in the midst of his surgery, viewing a large succession of diverse cases in quite short periods of time, will at once have his attention arrested when he comes across a possible case. Before qualification the student tends to look no further ahead than the next hurdle of examination, and the cases that he sees in the wards and in out-patient departments are valued by him according to whether he thinks they will turn up like a number at a roulette table during the next examination paper. He may well forget that a great deal of his future professional life will be spent in sorting out cases of early but serious disease from a whole mass of apparently trivial ailments, and that the lives of his patients are in his hands, to the extent of how soon he can arrive at an accurate diagnosis. Mr. Cope lays just emphasis on the large number of patients with actinomycosis whose condition is diagnosed only in its later stages and after months or years of illness and disability, during which they will have undergone a variety of useless treatment, before they finally encounter a clinician who recognizes the specific disease and its specific remedy.

Antibiotics, particularly penicillin, can bring about such a profound arrest of the infection that the general practitioner should regard it as a serious responsibility not to miss a single one of these cases among his own patients. This is the object of Mr. Cope's book. With an admirable clarity suited to the jaded intelligence of the general practitioner, of, it may be, a quarter of a century's work—and equally suited to the over-stuffed brain of the final-year student—he takes us over the natural history of the disease. He stresses the importance of recognizing the usual point of entry into the body via the decayed tooth, and points out the lesson of preventing actinomycosis by attention to the teeth. The illustrations of cases with the typical scarring of the neck should give such an impression that the general practitioner who has never seen a case ought to be able to say to himself when he meets one, "Surely this could easily be a case of actinomycosis?"

All the illustrations are beautiful, especially the coloured photomicrographs; and for those who learn better in rhyme there is a series of jingles reminiscent of student days. Everywhere through the book there runs the golden warning to the general practitioner that it is not sufficient to evacuate pus without searching for the organism responsible for its formation. And where the general practitioner has, as he should have as a right, open access to the local pathological department, examination of pus from any doubtful sinus would reveal actinomycosis in its early stages and save much misery and unnecessary suffering to his patients.

G. O. BARBER.

ANTIBIOTICS

Antibiotics. A Survey of Their Properties and Uses. Published by direction of the Pharmaceutical Society of Great Britain. (Pp. 290; 24 illustrations. 25s.) London: Pharmaceutical Press. 1952.

The Physician's Guide to Chemotherapy. By Peter N. Swift, M.R.C.P.Lond. (Pp. 176. 15s.) London: H. K. Lewis. 1952.

The volume on antibiotics published by the Pharmaceutical Society is called a second edition of the book on penicillin published from the same source in 1946, but it is in fact almost entirely a new work. It has five authors and at least a dual public, of whom the pharmacist gets 76 pages on preparations (and is chiefly interested in 12 on stability), while the practitioner will turn chiefly to 32 on "experimental background" dealing with the theory of antibiotic therapy and 34 on clinical use. Earlier chapters on history, manufacture, chemistry, and assay methods are of interest to both and to other types of reader as well. There is also a chapter on veterinary uses.

Taken as a whole, this book is the best source of information on its subject within the compass of a small volume which has appeared in this country, and it is very fully documented. In the clinical section there is a useful table stating in precise terms the sensitivity of important bacteria to all the main antibiotics. Some additions might be made to it with advantage; gonococci and meningococci are omitted, no entry for penicillin appears against any gram-negative bacillus (although two species listed deserve it), and there is no entry for chloramphenicol against either staphylococci or *H. influenzae*. The last is extremely sensitive to this drug, and it is doubtful whether chloramphenicol should be described as less effective than aureomycin in the treatment of *H. influenzae* meningitis, particularly since it traverses the blood–brain barrier more freely than any other antibiotic. Penicillin is recommended for sinusitis without qualification in regard to the causal organism, and for breast abscess regardless of the fact that in maternity units this condition is now almost invariably due to a penicillin-resistant staphylococcus and thus requires aureomycin or

terramycin. Apart from these minor disputable points, this section is an informative and reliable guide to antibiotic therapy, and, like the rest of the book, gives a good account of fundamentals.

The author of *The Physician's Guide to Chemotherapy* tries in only 176 pages to discuss from the practitioner's point of view, not only antibiotics, but sulphonamides and allied compounds. Of these pages, 61 are taken up in describing drugs and their properties, and the remainder in prescribing rules for their use in diseases classified mainly on a regional basis. The descriptions in the text of the range of activity of different antibiotics are rather vague, and a table of normal sensitivities would be a useful addition. The final "Table of antibiotic activity," placing drugs in order of merit for the treatment of infections due to specified bacteria, does not serve quite the same purpose, although it is a useful guide. As another example of rule-of-thumb prescription, penicillin or sulphadimidine is recommended for acute bronchitis regardless of its causation, yet some bacteria causing this condition are insensitive to either drug. The reader will be rather puzzled by two apparently incompatible statements about the effect of penicillin in diphtheria, and will, one hopes, realize that in the tables on pp. 15 and 21 "mg. per cc." and "mg. per ml." are both mistakes for mg. per 100 ml. Within the limits set by the small size of the book, the author achieves much of his purpose. He discusses briefly many uncommon diseases as well as those of everyday experience.

L. P. GARROD.

INFANT DEVELOPMENT

Infant Development: The Embryology of Early Human Behaviour. By Arnold Gesell, M.D. (Pp. 108; illustrated. 21s.) London: Hamish Hamilton. 1952.

In his latest book Dr. Arnold Gesell tells the story of a new study by cinematograph of the development of the human infant and links it by special diagrams to foetal development. He repeats, therefore, to some extent what was set out in a book published in 1945 on *The Embryology of Human Behavior.* (Incidentally, it may be remarked that the spelling seems to vary from dust-cover to contents.) A chapter is devoted to still photographs from the film, but the selection is not full enough for the beginner, and in some instances will be over-familiar to those who have studied earlier books from Dr. Gesell and his collaborators. Then come nine chapters of comment ending with a brief essay entitled "A Developmental Philosophy." There is much of interest here, for the whole idea of the Gesell school is full of interest. But apart from creating a desire to see the film —called "a conceptual type of motion picture"—this new volume was slightly disappointing to the reviewer. The text runs to fewer than 100 pages, though there are 20 pages of useful information in the appendices. The meaning of some sentences is not altogether clear, illustrating the difficulties of a so-called common language. Now that Dr. Gesell can look back over 35 years it would be interesting to have his mature views on development at some length in simple language and without illustrations. Such a book should not refer to previous studies, and it should have an entirely original text.

ALAN MONCRIEFF.

MEDICAL STATISTICS

Elementary Medical Statistics: The Principles of Quantitative Medicine. By Donald Mainland, M.B., D.Sc., F.R.S.Ed., F.R.S.C. (Pp. 327; illustrated. £1 5s.) London: W. B. Saunders Company. 1952.

Professor Mainland has written a book for medical students which is more than an elementary introduction to statistical methods. He has attempted to show the relevance of modern techniques to all forms of medical investigation. He is most successful in the laboratory field, where his enthusiasm for small-sample techniques and his own experience are particularly relevant. Much of the book, in particular Chapter II, "On Looking at Evidence," would be read with advantage by all medical students, whatever their future career. This universal appeal is aided by the choice of examples, which are used freely throughout the book to illustrate each point. They are drawn from a wide variety of medical problems and are, in general, worked out in sufficient detail to enable the student to follow each step. However, the setting out is often poor and makes the reader's task unnecessarily difficult.

It is a pity that, with so much in its favour, the work is spoilt by uneven writing. The earlier chapters proceed at a reasonable pace for a beginner. Indeed, the discussion of the exact binomial distribution in Chapter III is almost too drawn out, particularly as most workers find the normal curve approximation is an adequate substitute. Later the pace quickens, so that the last two chapters rush the reader through a bewildering assortment of subjects, many of which would require far more discussion and illustration before they could mean anything to a novice. The author is at fault in two statements. It is not a valid measure of a prophylactic immunization to compare, as he suggests, the incidence of the disease in two areas, in one of which the vaccine on trial is used and in the other a placebo, however carefully these areas are chosen. Further, the expectation of life recorded in a life table for a given year is not the mean age at death of the people who died in that year.

The binding and printing have been kept simple in order to reduce the cost, but at 25s. a more durable board cover which would stand up to frequent use might have been expected.

J. KNOWELDEN.

POETS' INFIRMITIES

The Infirmities of Genius. By W. R. Bett. (Pp. 192. 18s.) London: Christopher Johnson. 1952.

Has the sickness of poets any relation to their creative activity? Is there a physical and mental price to be paid for the single-minded passion which the practice of poetry demands? Would Keats have conquered his tuberculosis if he had not been a poet, and would his name have been "writ in water" had his health been good? Dr. W. R. Bett, in this book of biographical studies of literary genius, appears to answer such questions affirmatively, although he would hardly claim for these essays, as his publisher does, that, "in every case, infirmity is shown to have a perceptible effect upon the shaping of thought and the development of genius." Indeed, his most precise suggestion on the effect of disease on literary genius occurs in the essay on Keats, where he writes, "It is tempting to speculate that the toxins elaborated by the tubercle bacillus paralyse the highest critical faculties localized in the cortex of the brain, with the result that the so-called 'vegetative' faculties such as imagination, rhythm, colour sense, and possibly poetry are now free to act." Such speculations raise more questions than they answer, and Sir Russell Brain (*British Medical Journal*, 1949, **2**, 1427), writing on the relation of literary genius to mental disorder, admirably stated his opinion that "clearly no man is a great creative artist because he is a psychopath, but our subjects surely show that, given the necessary qualities of mind, more than one process which the psychiatrist calls pathological may be an essential element in the virtues of genius."

If it appears improbable that the rheumatic fever of Robert Burns, the syphilis of Baudelaire, the epilepsy of Swinburne, and the hypertension of Balzac had a dynamic relationship to their literary performances, there can yet be little doubt that the talipes of Byron and the kyphosis of Pope embittered their minds and envenomed their poetry. Whatever one's opinion of these problems, these essays make fascinating reading. A doctor who loves literature and can write finely is well endowed to understand and explain the personality of the creative artist. Dr. Bett has these qualities and in this book he makes splendid use of them.

D. V. HUBBLE.

BRITISH MEDICAL JOURNAL
LONDON
SATURDAY JANUARY 10 1953

AUTONOMIC CONTROL

Psychosomatic medicine has not become a separate specialty in this country, and even the more moderate claims of its devotees have met considerable criticism or disbelief, perhaps because the physiological mechanisms involved have been little explored. Nevertheless, there can be few clinicians who are unaware of the role which emotional disturbance can play in the precipitation and aggravation of somatic disease, and especially is this so in diseases of the skin. But, though emotional disturbances are common, they do not usually cause skin lesions, which are relatively rarer. Therefore the skin has been considered to be particularly susceptible to emotional disturbances in these cases. The techniques of treatment employed—abreaction with ether, carbon dioxide, or methylamphetamine—are physical methods which affect the autonomic nervous system, and the psychotherapy given at the same time may be small. It is possible to infer therefore that pathological changes in the skin may be primarily due not to emotional disturbance but to faulty reaction in the mediating vegetative nervous system, somewhere between higher brain centre and peripheral vessels. Shorvon and his coworkers[1] appear to have recognized this possibility, as they noticed that some successful results were obtained by "non-specific abreaction."

The commonest clinical entity in which there is nervous disturbance of vegetative function is manic-depressive psychosis. Here there are marked temporary alterations in peripheral autonomic functions: in the circulation, in sweating, in gastro-intestinal and sexual activity. At the same time there is a dramatic and shattering disturbance of mental function, much more devastating in its effect than any physical disturbance which can reasonably be attributed to psychic inefficiency. While this psychosis has been firmly ascribed to an inherite nervous instability,[2] the actual site of the disturbanc has never been demonstrated, though it is reasonat to place it within the higher autonomic centres. Ii leed, typical manic symptoms may occur in close association with small lesions confined to the hypothalai us.

What is now known of central autonomic regulation? Considerable work has been done in investigating the nervous control of the blood vessels.[3] Abrupt alterations in vascular tonus, nervous in origin, can occur in response to various stimuli—pain or cold, a sudden loud noise or "startle" stimulus, or even a purely psychological change. Because of the bilaterality and uniformity of response and because of the marked adaptation that follows repetition of a given painful stimulus it was for long thought that the resulting vascular contraction was entirely central in initiation and depended on the integrity of thalamic and hypothalamic structures. Carmichael[3] has demonstrated, however, that these responses can be obtained from areas innervated by an isolated section of spinal cord. More recent work[4] has shown that this local reflex can be demonstrated in normal subjects, and it is clear therefore that some autonomic regulation takes place at the level of the spinal cord.

Some integration of autonomic functions also takes place in the medulla. But the more complex control of the sympathetic and parasympathetic nervous system is vested in the hypothalamus. Tumours in this region produce pathological sleep or hunger, obesity, amenorrhoea, hypertension, or, occasionally, paroxysmal autonomic storms which Penfield[8] has termed diencephalic epilepsy. Recent work on hypothalamic control over both the autonomic nervous system and the endocrine glands was reviewed not long ago.[5]

It is, however, only during the last few years that the role of the cortex in controlling autonomic functions has been fully recognized, and its importance has been underlined by the successful treatment of mental illness by neurosurgery. While leucotomy is often helpful in mental states in which psychological tension is the main aetiological factor, it is becoming clear that it has a specific effect chiefly in depressive illness, especially of the involutional type, which is often characterized by impairment of autonomic function. The anatomical studies of Le Gros Clark in this country, and the exploitation of the technique of physiological neuronography by McCullough and others in the United States, have indicated the extensive nature and complexity of the cortical and subcortical areas which are thought to be involved both

[1] *British Medical Journal*, 1950, **2**, 1300.
[2] Slater, E. O., *Proc. roy. Soc. Med.*, 1936, **29**, 981.
[3] Carmichael, E. A., *Brit. med. Bull.*, 1950, **6**, 351.
[4] Elithorn, A., Piercy, M. F., and Crosskey, M. A., *J. Neurol. Psychiat.*, 1951, **14**, 209.
[5] Harris, G. W., *British Medical Journal*, 1951, **2**, 559, 627.
[6] Meyer, A., and McLardy, T., *J. ment. Sci.*, 1949, **95**, 403.
[7] *Res. Publ. Ass. nerv. ment. Dis.*, 1950, **29**, 775.
[8] *The Cerebral Cortex of Man*, 1950, New York.

in the control of autonomic responses and in the elaboration of emotional feelings. It is now known that the temporal horn and the posterior orbital surface of the frontal lobe are closely interconnected both directly by the uncinate funiculus and indirectly through the hippocampal-fornix system. Again, the temporal and the frontal regions are in intimate relation with the hypothalamus. Similarly the cingulate gyrus, which is believed also to subserve autonomic function, is connected with the hypothalamus by way of the thalamic nuclei. It is the dorsomedial nucleus of the thalamus which characteristically shows secondary degenerative changes following successful leucotomies,[6] and it is now generally accepted that this region is an important link between subjective emotion of cortical origin and autonomic function controlled by the hypothalamus. Unfortunately direct evidence, from stimulation in man, of cortical areas which influence autonomic function is still very scanty. Livingstone[7] has demonstrated changes in blood pressure and in the respiratory rhythm following stimulation of the posterior orbital region, and Penfield and Rasmussen[8] have elicited autonomic responses from the lower end of the motor cortex and from the subjacent island of Reil, an area that is anatomically closely related to the posterior orbital region mentioned above. Other workers, by varying the rate and strength of stimulation, have succeeded in producing lacrimation from areas that customarily produce eye movements, and salivation from the same area as movements of mastication. Autonomic responses elicited by this type of stimulation are crude, as are somatic motor responses to stimulation. Probably many regions of the cortex are in fact capable of originating an autonomic response, but the adequate stimulus is likely to be a complex and specific pattern of neuronal activity, and it is not surprising that even in the conscious subject large cortical areas remain unresponsive to electrical insults, however carefully the voltage, frequency, and duration of the stimulus are controlled.

Present knowledge thus supports the hypothesis that there is a primary autonomic motor area similar to the motor cortex, closely related to it anatomically, and probably extending down through the Sylvian fissure on to the posterior orbital surface. Just as complex motor actions are organized in association areas relatively unresponsive to stimulation, so then are there likely to be areas predominantly concerned with the elaboration of the autonomic effects accompanying emotion. Some of these areas are almost certainly in the anterior regions of the frontal lobes.

SCIENTIFIC STUDY OF DELINQUENCY

As psychology has ventured into the sphere of conduct and behaviour, at one time considered to be the preserves of free will, problems of delinquency have come under the scrutiny of scientific investigation. The oft-debated question whether crime is a disease, which Samuel Butler brought to the fore in *Erewhon*, will now provoke the answer that sometimes it is and sometimes it is not. And scientific study enables us to distinguish types of crime not only according to the nature and seriousness of the offence, as the law does, but according to the cause of it in the individual and society. The emphasis has passed from study of the delinquency to study of the delinquent, from the crime to the criminal, from his act to the motives for his act.

The mental defective or epileptic who commits acts of violence is in a different category, for instance, from the man who commits the same crime because he has been brought up with no respect for social values or because he prefers to earn his living that way. Psychological treatment is not the primary need of the latter. In a third category, and one less well known to the public, are those people who commit offences under the influence of an obsessional compulsion. They are often of otherwise good character. The schoolmaster and the magistrate are often baffled by the discovery that thefts from boys' lockers have been committed by the most conscientious and scrupulous pupil, and the public is usually scandalized when it learns of the homosexual lapses of some highly respectable man. Such delinquents are themselves bewildered and embarrassed by these propensities in themselves, so opposed to their normal character and so contrary to their will, and on discovery they are filled with remorse. They may desire punishment to expiate their crime, but this has no lasting effect. Such conditions can often now be cured or at least alleviated.

Less understood, and for that reason worth careful attention, is the group of "reaction delinquents." These, unlike the last group, are defiant in their delinquency but yet, like the last group, often come from good homes. Typical is the man who, unwanted and unloved as a child, reacts by suppressing all desire for love and becoming anti-social, resentful, and rebellious. In early years he is the difficult child, in adolescence he may become the delinquent, and in later life the criminal. He has no remorse, and remains defiant. Punishment, instead of improving his condition, makes him worse, for it increases his resentment and sense of grievance. The more these

reaction delinquents are punished the more crimes they commit, and it is these that account for many of our so-called recidivists. Only by winning the confidence of such people, discovering the cause of their delinquencies, and eradicating them by suitable psychological treatment can they be restored to a social way of life, and the results of such an approach are gratifying.

Clearly to treat these four categories of criminals in the same way will bring success only in those cases where the treatment is appropriate, and failure in most. It is in distinguishing the different types of delinquents that the scientific approach is most useful, and magistrates on the whole welcome it. Britain is fortunate, and probably unique, in having an institute devoted entirely to this subject—the Institute for the Study and Treatment of Delinquency, founded 20 years ago. Though the National Health Service has taken over its clinic, now known as the Portman Clinic, I.S.T.D., the institute remains independent, and both bodies work in close co-operation. The institute's research work is conducted, under a scientific committee and an honorary director, by paid research workers. Its educational work covers lecture courses for magistrates, probation officers, those taking social science courses in the University of London, and week-end residential courses for social workers and others. A feature of the institute has always been co-operation between members of the medical and legal professions, and its latest annual report[1] shows that the executive committee consists of three lawyers, a psychiatrist, an educational psychologist, and a chartered accountant—a breadth of interest that should disarm those who are ever ready to resent the intrusion of psychology into criminal affairs. About half the patients referred to the clinic come from the courts, while in some cases prisoners voluntarily apply for treatment after serving their sentence. Perhaps a sound guide to the value of the work is that the institute and clinic retain the confidence of the courts, and magistrates continually make use of the clinic for diagnostic reports and for treatment under probation.

RADIOLOGY ON POINTS

"This memorandum sets out a new method of recording the work undertaken in diagnostic *x*-ray departments, which will come into use on January 1, 1953." Thus begins circular 94121/1/14 issued by the Ministry of Health on December 13, 1952, and many radiologists take strong objection to it, not least because once again the Ministry of Health appears not to have sought co-operation and agreement with the profession. Three years ago the Nuffield Provincial Hospitals Trust looked into methods of estimating the work done in hospital radiodiagnostic departments. A subcommittee of the B.M.A.'s Radiologists Group Committee joined with the Trust in this inquiry, and it was eventually decided to try out in 1952 the "unit" system which is the subject of the Ministry of Health's edict in circular 94121/1/14. It was arranged that at the end of 1952 the Radiologists Group Committee of the B.M.A. should receive a report on how the system had worked, so that it could consider its worth and possible modifications. The Ministry, in what might be termed its *folie circulaire*, could not even wait until the end of the year. Out it came with its circular instruction on the unit system of estimating work done in diagnostic *x*-ray departments. Apparently the Ministry had taken this scheme from the Nuffield Trust, which had had it from the Radiologists Group Committee, and went into print with it *without any consultation with the B.M.A. or its Group Committee*. No doubt the Ministry's officials wanted to get their desks cleared well in advance of the Christmas holidays, but, if we may quote from a recent leading article[1] on the Ministry's unilaterality—"This is an excellent example of how not to do things." Those responsible for the work of diagnostic *x*-ray departments will have every justification in refusing to obey the instruction in circular 94121/1/14.

And what of the system itself? You can have a "gallbladder, plain" for 1 unit, "urinary tract, plain" for 2 units, or "Smith Petersen pin, insertion of and similar procedures" for 8 units. At the end of the year the units will be added up, and then calculated in terms of radiologists and their technical assistants, no doubt also in units. Apparently the system—in its trial period—has worked satisfactorily in some hospitals. But the criticisms levelled against it by Dr. J. W. D. Bull in this week's *Supplement* (p. 11) have great cogency; and he speaks with the authority of the man doing the job. There is, as he points out, all the difference between *x*-raying the spine of a slim adult and that of an obese, incontinent, and paraplegic patient lying in plaster. The time taken for a ventriculography (10 units) may vary from 10 minutes to two hours. In short, human beings just will not fit into a Ministerial sliding scale. And in the background to all this is not there an element of distrust? Cannot the Ministry trust hospital staffs to do their work with a sense of responsibility, and not to ask for more staff than is necessary? If medical men are instructed by the Ministry that *x*-raying "spine, more than one area" is worth only 4 units of work,

[1] *British Medical Journal*, 1952, 2, 1140.

is there not a suggestion that, following the example of the bricklayer, they will end up by downing tools on the completion of the fourth unit? Is this really medicine in Britain to-day? It is to be hoped that the Ministry of Health will have second thoughts.

OUTBREAKS OF SKIN DISEASES

With an easy grace Professor R. Cruickshank in his Malcolm Morris lecture, published in our opening pages, takes three or four skin diseases from a "large and fascinating field" to illustrate the principles and practice of epidemiology. Malcolm Morris was one of the earlier generation of British dermatologists and, like his colleagues and his immediate successors, was a great teacher, a man of learning with broad vision and wide interests. Apart from dermatology he contributed seriously to problems in tuberculosis and public health, and he was keenly aware of the significance of general medical problems as reflected in the skin. Dermatology has had a preview of many of the problems that have subsequently become prominent in general medicine, as in syphilis and tuberculosis and, more recently, in the sarcoidoses and the reticuloses and what are at present termed the collagenoses.

It is rather surprising, therefore, that dermatology has not received more emphasis in the British medical curriculum. On the Continent and in America it assumes the important place in medical affairs that might be expected of the study of an organ which is so readily observed and investigated. And surely a student or practitioner who can recognize the principles and pursue the essentials of medical practice in dermatology is well equipped to study and understand the disorders of other organs. British medicine has above all things been strong in the clinical field, and the neglect of dermatology must lead to a loss of opportunity for the clinician. It is almost a corollary that medical mycology has also received little attention in this country, as Cruickshank points out, since most of our significant mycoses are skin affections. Yet valuable pioneer work was done in medical mycology by British dermatologists in the earlier part of the century.

From many independent studies it would seem that impetigo of bullous type, including pemphigus neonatorum, is staphylococcal in origin, and Cruickshank shows how the source of infection can be traced by the study of coagulase production and phage-typing. In the adult it would seem often to be an autogenous infection from a nasal reservoir, the skin lesions being started by injury. In cases of pemphigus neonatorum, infection may come from nurses or other attendants or from the noses of other infants, but not usually from the mother of the infant. Tropical impetigo shows many of the features of pemphigus neonatorum and is also due to the staphylococcus, profuse sweating predisposing to the infection. Streptococcal impetigo, characterized by "stuck-on scabs" and often associated with lymph-node involvement, is apt to become epidemic, and appears to bear some relationship to the incidence of scarlet fever. Like that disease, it is milder than formerly and is disappearing.

For some reason impetigo has not been satisfactorily reproduced experimentally with either staphylococci or streptococci, though the contagious character of the disease is evident. It is at times difficult to find cases of impetigo in dermatological departments to demonstrate to undergraduates, though in some clinics where the disease has reappeared a penicillin-resistant organism is usually present. Cruickshank warns against the use of penicillin cream for this very reason. Since 50% of the population carry dangerous staphylococci in their noses it would seem desirable to practise nasal hygiene as seriously as we practise oral hygiene.

MEDICAL CARE OF THE SCHOOL-CHILD

While two Ministries and the local education authorities divide between them the medical care of school-children there will continue to be considerable overlapping in the services provided. The child with otorrhoea may be looked after in the family doctor's surgery, in the minor-ailment clinic, or in the out-patient department of a hospital. The citizen pays for all these services, and his representatives in the Commons or council chamber are indifferent about which service the mother of his child decides to use. This is an extravagant redundancy of medical care, and it would be easy for a director of the child medical services, if one existed, to insist that the remedial activities of the education authority should be limited to the care of teeth, the correction of disturbances of behaviour, and the cleaning of verminous heads. But, though an easy decision, it would deny the historical reasons for the growth of this child-care, exacerbate dying controversies, and might for some time diminish efficiency of treatment. Changes will and must be made in this and many other directions, but it is wiser to allow them to evolve gradually and locally without seeking to impose on them a universal pattern. Meanwhile, the three agents of these services—in general practice, in the school medical service, and in the hospitals—should make their own work as careful and instructed as possible, seeking co-operation with their colleagues and being ready to proffer the courtesies which a common professional interest demands. Our columns have recently shown[1] that considerable thought is being given to the problems of co-operation between these various agencies, and if they have also shown that individuals, particularly the general practitioners, are jealous of their responsibilities towards the child, this attitude is to be welcomed and not deplored. The now greatly improved training of medical students in paediatrics reinforces the general practitioners' claim to an important share in the medical care of children.

Meanwhile the latest Report[2] of the Chief Medical Officer of the Ministry of Education clearly indicates the directions along which the school medical service will continue its remarkable work. It will seek to improve the physical education of the school-child, and

[1] Hinden, E., *British Medical Journal Supplement*, 1952, **2**, 103; see also correspondence, ibid., pp. 127, 128, 136.
[2] Report for 1950–1 of the Chief Medical Officer, Ministry of Education. H.M.S.O. 5s.

there is an admirable chapter in the report on the place of swimming in the school curriculum. It will encourage hygiene in the schools, and such subjects as sanitary towels, clean canteen food, and infested heads are here discussed. It will give increasing attention to the education of physically and mentally handicapped children, and a survey of the needs of these children occupies one-third of the report. This section contains some outspoken criticisms on "open-air" schools, which show that the school medical service is ready to correct its own errors and inadequacies. There is an excellent chapter on the growth and nutrition of the school-child which would do credit to a textbook of paediatrics, and it illustrates the opportunities for group research which exist in the school medical service.

The report occasionally reveals tendencies which will not receive unanimous approval. Some will not regard the electrocardiograph as a useful apparatus in the central school clinic. Many will question the value of applying the laborious technique of Sheldonian somatotyping to children. All must deplore imposing on school medical officers the duties of medical examination of entrants to training colleges and of local-authority employees for their fitness for superannuation—a departure which the report unctuously accepts as a new responsibility for the staff of the school medical service and an additional use for the school clinic. Whitehall does not hesitate to criticize the local authorities; here is an opportunity to practise a little self-criticism.

THE TOXICITY OF IRON

Many of the elements and chemical substances essential to the body can prove to be poisonous if administered in excessive quantities. Iron is an essential component of many respiratory pigments, including that present in the largest amounts in the body—namely, haemoglobin. Certain forms of anaemia seem to result from a defective supply of iron to the tissues, and in some types the defective supply of iron may be coupled with an increased rate of loss or destruction of blood. It is not always clear where the fault may be in many conditions of microcytic or "iron deficiency" anaemias, but the condition may often be improved or corrected by administering iron.

Iron is not normally absorbed very readily from the gut, and most of that taken by this route is excreted. Work done in recent years suggests that there is some physiological barrier or block regulating the intake of iron from the gut. In some cases of anaemia this block may apparently prove so effective that adequate therapy by the oral administration of iron salts becomes impossible, and patients remain anaemic despite large doses of iron by mouth. On and off for many years different preparations of iron have been given by injection. It often proved to be effective by this route, but the hazards of administering the metal prevented the general introduction of parenteral iron therapy: to quote one textbook, it is "as toxic as arsenic" when given intravenously. Since the report by Nissim[1] in 1947 of the safe and successful use of a saccharated iron preparation, intravenous iron has been given quite widely in this country. And many cases of anaemia have responded dramatically and without reaction to the treatment.

There have been a number of reports of toxic reactions to intravenous iron, and a further two cases were described in this *Journal* last week by Librach.[2] He discussed at some length the possible aetiology of the condition. Not all the other cases described resemble his, and, apart from vasomotor disturbances during or immediately after the injections, many of the reactions have followed several hours later. One writer compared them with the response of cases of paroxysmal haemoglobinuria to cold and suggested it was a sign of increased activity in the haemopoietic tissue due to the administration of the iron. Embolism might be the cause of the severe central disturbances, though a similar condition has been described after excessive oral administration of iron salts.

Although some anaemic people seem unable to absorb iron by mouth, small children seem to be able to do this all too readily. There have been a number of reports over the past 10 years of serious and fatal poisoning among small children who have found the brightly coloured pills of ferrous sulphate fatally attractive. Smith[3] in describing some of these cases suggested that death may be due to "shock" and that the shock results from an excessive formation of the vasodepressor material of Schorr, Zweifach, and Furchgott.[4] This material, which can be isolated from tissues, has been shown to be identical with ferritin. Ferritin is a protein complex formed with iron, and its formation is believed to be a factor controlling iron intake from the gut. Smith suggests that the administration of large doses of iron leads to an excessive production of ferritin and that its release into the circulation produces shock. Ferritin is also formed in the tissues from the serum iron, and as this remains high too, as a result of continued absorption from the large amounts in the stomach, the condition is aggravated by the continuous formation of ferritin.

We might suppose that the signs of poisoning by intravenous and oral iron would be the same if there were any substance to this theory or if the toxic responses to intravenous iron were in fact due to the iron. Unfortunately it is impossible to find any features of the two types of poisoning which are commonly shared. Some of the toxic properties of saccharated iron may not be due to the iron at all but to the sugar,[5] and some of the reactions may be due solely to its properties as an irritant. The importance of the dose in determining the onset of a toxic response is emphasized by Librach's second case. It is possible that reactions occur only when previously unsaturated serum protein becomes saturated with the added iron. Yet in one large series of cases[6] it was found that gradually *increasing* doses appeared to prevent reactions. Although we may know a good deal about the physiological behaviour of iron

[1] *Lancet*, 1947, **2**, 49.
[2] *British Medical Journal*, 1953, **1**, 21.
[3] *J. Path. Bact.*, 1952, **64**, 467.
[4] *Science*, 1945, **102**, 489.
[5] Robson, J. M., and Keele, C. A., *Recent Advances in Pharmacology*. London: Churchill. 1950.
[6] Wilkinson, J. F., and Slack, H. G. B., *Lancet*, 1949, **1**, 11.

when it is intimately bound to complex proteins as in haemoglobin, the way in which the element and its simple salts may disturb physiological function remains obscure.

SOFT-TISSUE RADIOLOGY

Radiography, first used in medicine for the demonstration of fractures and foreign bodies, progressed rapidly to the use of artificial contrast media such as the barium meal. To-day the more complex contrast examinations are commonplace and have, for example, removed the diagnosis of cardiac anomalies from the realm of guesswork. The true soft-tissue examination, without artificial contrast, is not so well known outside radiological departments. The problem is primarily a physical one: all soft tissues absorb *x*-radiation to almost the same degree, the sole exception being fat, which is more transparent and therefore more easily demonstrated, and which may serve to delineate any structures it surrounds. The kidney, for example, is seen in the plain abdominal film because it is surrounded by perinephric fat. A variety of techniques have been suggested for studying the soft tissues, which, because they are all so alike to *x* rays and so much less radiopaque than bone or barium, require very precisely adjusted radiography. They demand considerable skill in the radiographer and the results are little seen in the medical press, for they require an expert illustration department for their reproduction, and very good blocks printed on good paper.

The position is surveyed and admirably illustrated in Frantzell's[1] recent monograph. He has applied the method to the extremities, and the paper has several fine illustrations of dystrophic and oedematous muscles. Varicosities of superficial veins may be seen as worm-like tortuosities in the subcutaneous fat. Shiers has been able to show the value of soft-tissue technique in the examination of tumours of the larynx. Yet another use of soft-tissue radiography is in examining the placental site and the form of the pregnant uterus, which is clearly shown by Reid's[2] technique: the diagnosis of placenta praevia becomes a precise matter. Greenhill,[3] making a plea for the wider use of placental radiography, considers that if, for all his patients with vaginal bleeding, caesarean section had been routinely employed without radiological sorting the operation would have been unnecessarily performed in 88% of cases.

Uncalcified tumours may often be demonstrated by slight density differences, which are lost on over-penetration by the rays. Mammography—radiology of the breast, either plain or after duct injection—has been recommended by Lame and Pendergrass,[4] and intramuscular or superficial lipomata, by reason of their lower density (greater transparency), are among the simplest tumours to demonstrate. Gas inclusions in the tissue planes are also readily shown as diminished density, and surgical emphysema is a common finding after thoracotomy. The gas formed in anaerobic infection is very obvious on a film, but experience during the 1939–45 war showed the unwisdom of waiting for this sign, clear though it may be.

There is a trick which, though not true soft-tissue radiography, is of value in some plastic surgical work. In addition to the film placed normally between intensifying screens ready for the exposure, another sheet is placed in the cassette, outside the screens. This sheet of course will be grossly underexposed, and in a lateral view of the face only the soft outline will record; this film will register exactly with the normal one, and the facial contours may be accurately related to the skeleton.

Any description of soft-tissue radiography is inclined to become a catalogue, for the method is not a watertight compartment. It is an application of the basic physical principles to special cases, demanding only an intelligent use of available equipment. It is surprising that such applications are not more widely requested.

[1] *Acta radiol., Stockh.*, 1951, Suppl. 85.
[2] *Brit. J. Radiol.*, 1949, **22**, 557.
[3] *Principles and Practice of Obstetrics*, 1951, 10th ed. Saunders, Philadelphia and London.
[4] *Radiology*, 1947, **48**, 266.

THE NEW YEAR HONOURS

The list of medical men awarded honours in the New Year, published elsewhere in this issue, contains names known the world over and names whose habitation is more locally confined. All have deserved well of their fellow men, and their public recognition gratifies the profession to which they belong. Outstanding as a man of science and a surgeon, Professor Wilder Penfield enters the Order of Merit, limited in numbers to 24, the two other medical members of which are Dr. E. D. Adrian and Sir Henry Dale. Professor Penfield was born in America and subsequently became a naturalized Canadian citizen. Before and after the 1914–18 war he was a Rhodes Scholar at Oxford, where he was a pupil of Sir Charles Sherrington. The titles of his principal publications—*Cytology and Cellular Pathology of the Nervous System*, *Epilepsy and Cerebral Localization* (with T. C. Erickson), *The Cerebral Cortex of Man*—show the nature of his contributions to medical science and neurosurgery. The importance of the work of the medical branch of the Colonial Office is fittingly recognized by the bestowal of the K.C.M.G. on Dr. E. D. Pridie. Surgeon Vice-Admiral K. A. I. Mackenzie, Medical Director-General of the Navy, and Lieutenant-General F. Harris, Director-General of the Army Medical Services, are both awarded the K.B.E. The conferment of a knighthood on Mr. V. Zachary Cope and on Professor A. W. M. Ellis will give especial pleasure to their many friends and colleagues. Among the distinguished Australian doctors honoured this year is Dr. N. M. Gregg, whose work on rubella and congenital defects has been so notable. Medical research in this country is recognized by the bestowal of the C.B.E. on Professor R. A. McCance and, for his great administrative services, of a knighthood on Dr. A. Landsborough Thomson, Second Secretary of the Medical Research Council. Although the medical profession cannot claim him for their own, all who are music lovers will acclaim the award of the C.B.E. to Dr. Boyd Neel.

REFRESHER COURSE FOR GENERAL PRACTITIONERS

HEAD INJURIES—II. TREATMENT AND SEQUELAE

BY

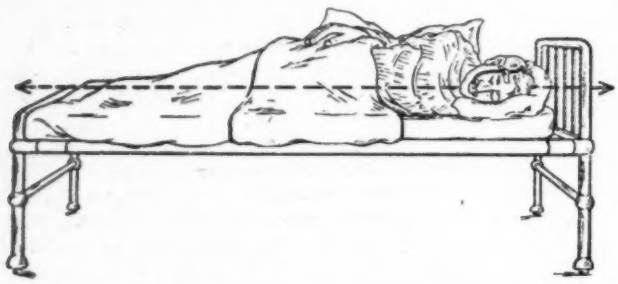
D. W. C. NORTHFIELD, M.S., F.R.C.S.

Surgeon-in-Charge, Department of Neurosurgery, London Hospital

Treatment of Closed Uncomplicated Injuries

After a head injury shock may be present, but it usually resolves spontaneously with simple methods of resuscitation—for example, warmth, rest, raising the foot of the bed, and tap-water per rectum. If it is severe and persisting, as shown by a low blood pressure (persistently below 100 mm. Hg systolic), rapid pulse, and cold and sweaty extremities, more energetic methods should be employed, including blood transfusion and a pressor drug such as methylamphetamine hydrochloride, 15 to 30 mg. intramuscularly.

While unconscious the patient should be nursed in the lateral position, and turned regularly from side to side every four hours; the skin over pressure areas should receive careful attention. The head should rest on a low pillow, and the body should be horizontal (see illustration). In this position the midline of the head will



lie in the same axis as that of the trunk, and the airway will be clear; respiratory secretions will flow from the uppermost lung along the trachea and pool with saliva in the lower cheek. The mouth and pharynx should be regularly cleaned out at intervals of, say, half an hour with a piece of moistened gauze held on forceps, or by suction from a motor pump through a large soft rubber catheter. When the patient is turned over on to his other side the lower lung takes the upper position and is then drained of secretions. In the event of there being excessive secretions (as may happen after a brain injury) the foot of the bed must be raised so as to take greater advantage of the action of gravity in emptying the respiratory pathways.

It is wrong to elevate the head and shoulders of an unconscious patient, in whom the coughing mechanism is usually defective, for this will increase hypostasis and embarrass respiration still further. It is also wrong to nurse the patient supine, for the tongue will fall back and obstruct the airway. Oxygen should be administered if there is any cyanosis; anoxia will aggravate cerebral damage. Pyrexia should be countered by removing bedclothes; if the temperature rises above 103° F. (39.4° C.) energetic measures must be employed—for example, covering the patient with only a damp sheet, playing an electric fan on him, administering ice-cold water per rectum. If unconsciousness lasts more than a few hours it is wise to give penicillin, 250,000 units twice daily, as a prophylactic against respiratory infection; if infection becomes established larger doses will be necessary, 1,000,000 units twice daily. The sputum should be cultivated and micro-organisms tested for sensitivity to other antibiotics and the sulphonamides. Pulmonary collapse may occur as a result of the plugging of a bronchus with viscid mucus and bronchoscopic aspiration may be necessary.

During the first few hours of unconsciousness special attention to fluid intake is unnecessary, apart from the treatment of shock. At the end of that time tap-water may be given per rectum. If consciousness has not returned within 12 hours food and nourishment must be given by means of a soft rubber tube (Jacques's oesophagus tube No. 9 English gauge) passed through the nose into the stomach. Great care must be taken to ascertain that the end of the tube is within the stomach and not in the trachea, for it can easily be passed into the trachea in a deeply unconscious patient. The safest test is to inject (or blow) air briskly down the tube while listening with a stethoscope placed over the stomach; unmistakable loud borborygmi will be heard as the air bubbles into the stomach. The tube may be removed once weekly for cleaning. As consciousness returns the swallowing mechanism must be carefully tested; a swab soaked in water is held between the lips and squeezed and the throat watched. If sucking and swallowing are satisfactory, teaspoonfuls of water may be given, and later a feeding-cup may be employed. If the fluid is not swallowed properly, or coughing occurs, the tests should be repeated daily until the nasal tube can be withdrawn. These details have been given, as proper nursing during the period of unconsciousness is life-saving.

When the patient is fully conscious he may take up whatever position in bed is found to benefit his headache; he may be propped up on pillows, lie flat, or the foot of the bed may be raised. There is no hard-and-fast rule about posture. He should be kept in bed, in a subdued light, free from distractions and noise until headache, dizziness, and nausea abate. As these symptoms subside he may be given increasing liberty to sit up in bed for meals, read, smoke, and receive visitors.

How long should he remain in bed? The best criterion is freedom from headache. When headache has been absent for a few days he may be allowed up for increasing periods of time. This graduated convalescence is continued, each few days bringing extra activity; if symptoms recur a previous stage should be resumed. When physically active, increasing exercise should be prescribed with a view to imitating as closely as possible the conditions of employment. Massage and exercises, and perhaps orthopaedic advice, will be necessary for residual limb pareses, and disabilities such as diplopia and dysphasia will need specialist advice.

Throughout, the patient should be encouraged in the belief that he will make a good recovery. Moral support and a cheerful outlook play a big part in the successful return to employment, and the doctor should never show any anxiety. On the other hand, a careful assessment of the patient's prognosis and honesty towards the patient are also necessary. In severe brain injury residual intellectual deficit or a physical disability may quite clearly prevent a patient from returning to his original type of employment. Expert help will be needed to make a proper assessment, and the patient should be told as soon as it is certain what the future holds for him. It is as important that he should not return to work for which the injury has rendered him quite unfitted as it is to avoid a neurosis, the result of introspection or of anxiety, that he cannot work when in fact

he is well able to. The experience of head injuries during the war has led to a lessening of the period during which patients are kept in bed. Too long in bed is apt to foster the patient's belief in the seriousness of his injury and to breed neurosis. Some cases need little or no rest in bed. Thus a patient who has been concussed and recovers consciousness in a minute or so may feel quite free from any ill effects, and, provided none appear, he need not be kept from his work. Or his headache may last but a day or so, and he might be allowed to resume work a few days later. Moreover, during the period of rest in bed enough activity must be allowed to prevent boredom.

Symptomatic Treatment

Restlessness may have an ascertainable cause—for example, a full bladder or thirst. In the early stages of compression a patient may exhibit much motor activity, perhaps the external evidence of headache, and this possibility should be borne in mind before prescribing drugs. Simple drugs such as potassium bromide and chloral hydrate (30 gr. (2 g.) of each per rectum, or 20 gr. (1.3 g.) of each by nasal tube) should first be tried. If an injection is essential soluble phenobarbitone, 3 gr. (0.2 g.) intramuscularly, or the more powerful drug paraldehyde, 2 to 5 ml. intramuscularly, are both safe.

Headache if severe may be the earliest symptom of compression, and the possibility of this complication must always receive attention before prescribing. Simple analgesics such as aspirin, 10 to 15 gr. (0.65 to 1 g.), or codeine (as tab. codein. co.), one or two tablets, usually suffice. Magnesium sulphate as a rectal infusion (6 oz. (170 ml.) of 50% solution retained for half an hour) and attention to the most comfortable position are helpful. The use of morphine should be avoided if possible, because of the risk in the early stages of masking the clinical evidence of brain compression. If it must be used, only a small dose (1/12 to ¼ gr. (5.4 to 11 mg.) by subcutaneous injection) should be given, and it may be repeated in half an hour if it is inadequate.

Insomnia may be controlled by the sedatives already recommended or by other preparations in common use.

Giddiness tends to subside spontaneously, particularly with graduated exercise, and rarely needs special drugs, though phenobarbitone in small regular doses, ½ gr. (32 mg.) thrice daily, may be given if necessary.

Radiography and Lumbar Puncture

An *x*-ray examination of the skull is unnecessary in the early stages of the care of closed head injuries: radiographs do not influence treatment, the manipulations of the head may interfere with the airway, and good definition cannot usually be obtained owing to the patient's restlessness. But if operation is to be performed for a fracture, depressed or open, good films are essential in order to provide the surgeon with precise information about the site and size of the fracture and the presence of driven-in fragments.

Lumbar puncture may be of value in several ways, but it is potentially dangerous during the first day or so; it is in this period that compression of the brain most commonly occurs, and a lumbar puncture may well aggravate that condition and precipitate the patient's death. For that reason it should be carried out only on the recommendation of the surgeon who will be called upon to operate. But if the diagnosis of compression does not arise the removal of small quantities of bloody cerebrospinal fluid may relieve headache and diminish physical excitement and delirium. If pyrexia is excessive or persistent lumbar puncture is advisable in order to detect the presence of meningitis, which may otherwise easily elude diagnosis.

Surgical Treatment

It would be inappropriate in this article to describe the various operations which may be necessary in cases of head injury. They are for the most part highly specialized procedures, and patients in need of these should be immediately transferred to the nearest centre where such facilities are available. In this category will be cases of brain compression (suspected or actual), of compound and of depressed fracture, and of extensive scalp laceration. Small scalp wounds, after carefully shaving the scalp over an area of at least 3 in. (7.6 cm.) from the edges of the injury, should be excised, removing all devitalized tissue but only the narrowest margin of skin edge, and exploring any subcutaneous pockets which may hide debris. The wound is then powdered with penicillin and a sulphonamide and closed with stitches.

Cerebrospinal rhinorrhoea resulting from fractures of the anterior cranial fossa often ceases spontaneously in a few days, and this may be hastened by keeping the head and shoulders raised, but this posture may be adopted only if the patient is conscious. If the leak of fluid persists or is severe operation may be advisable in order to close the dural tear with a fascial graft. If the fracture is extensive operation may be advisable in order to insure against intracranial infection at a later date. During the period of rhinorrhoea, and for several days afterwards, it is advisable to give a prophylactic course of penicillin, 250,000 units twice daily, and of sulphadiazine, 2 g. four-hourly for 24 hours and then 1 g. four-hourly.

Fractures of the facial skeleton may occur in conjunction with a head injury, and it is important that these should be recognized quickly, so that treatment may be initiated as promptly as possible. Either the upper or the lower jaw may be fractured, and a fracture particularly apt to escape detection on account of bruising and swelling of the eyelids is one involving the margin of the orbit. The eyeball may be displaced, and if unreduced this fracture may be the cause of permanent diplopia, quite apart from the disfigurement. Appropriate specialist treatment will be necessary.

Fractures of the middle cranial fossa involving the ear rarely need operation. The pinna and meatus are cleansed daily with 70% alcohol, insufflated with penicillin and sulphadiazine powder, and then covered with a sterile pad in order to diminish the risk of infection entering the middle ear from the exterior. The meatus should not be packed, but prophylactic penicillin and sulphadiazine should be given. If the ear is already the seat of a chronic otitis media, or if an acute infection develops, mastoidectomy may be necessary.

The Sequelae of Head Injury

The post-contusional syndrome embraces the minor symptoms which may persist for some time after a head injury—namely, headache, dizziness, and mild mental symptoms such as impairment of memory, inability to concentrate, apprehension, and emotional instability. To what degree these symptoms are due to organic damage or are psychogenic it is often impossible to determine, and so interwoven are the possible causative factors that the attitude now is to avoid any attempt at labelling. Obvious causes of psychological stress, such as the patient's natural anxiety for the care of his family, or about his symptoms or his future return to work, should be dealt with in an understanding and common-sense manner. Impending litigation may be a troublesome factor and often delays recovery by the anxiety it breeds.

As previously mentioned, proper assessment of the ability to work and of the appropriate work for the man are most important, and in serious cases may need psychiatric aid. A simple explanation of symptoms and disabilities is often of great help in fostering the patient's self-confidence. Intolerance of noise and of family activities usually subsides with sympathetic understanding. A doctor's letter recommending employers to allow shortened hours for the first week helps to bridge the period of readjustment. Headache of a mild degree may need simple analgesics, and reassurance is necessary in order to prevent the

patient from believing his brain is seriously damaged. Provided a proper period of initial rest and graduated convalescence has been carried out, the patient should be encouraged to ignore headache and persevere. Occasionally severe headache persists, and takes on all the characters of a migraine. It is best treated by those drugs which sometimes help in migraine. The following remarks of Cairns are pertinent: "Almost every patient who makes a full recovery from concussion suffers at a certain stage of his recovery from headache. . . . It is one thing to have a headache; it is another thing to complain about it."

If considerable structural damage of brain tissue has been sustained the residual sequelae may be severe according to the areas involved. Widespread contusion with much haemorrhage may lead to substantial loss of white and grey matter, demonstrable by encephalography as an enlargement of the ventricles and of the subarachnoid spaces over the cortex. Or the damage may be restricted to the frontal lobes as a bilateral polar contusion, often associated with bilateral anosmia. In these patients there is usually a quite obvious intellectual deficit, which may improve somewhat with re-education, but which is permanent to a greater or less degree. Return to employment will depend upon the size of the deficit and the nature of the available work. Hemiparesis will continue to improve, with lessening speed, over several months, but severe degrees of weakness and spasticity may be a permanent disability. Disturbances of speech, of reading, and of writing may improve with special training, but a permanent residuum is likely.

Recovery from cranial nerve damage varies according to the individual nerves affected. Anosmia, whether partial or complete, is usually permanent from the time of injury. Similarly, damage to the optic nerves and chiasma rarely shows any improvement. In paralysis of the extrinsic muscles of the eyeballs there is a strong tendency to recovery, although final recovery may be slow and orthoptic exercises or corrective operations may be necessary. Damage to the branches of the trigeminal nerve is rarely permanent. Facial paralysis usually clears up; it may be advisable to seek the advice of an aural surgeon about the feasibility of exploring the canalis facialis, in order to relieve pressure on the nerve or to graft it. Hearing may be recovered, although if the auditory nerve is damaged deafness is likely to be permanent.

Epilepsy is a serious sequela which may develop early or late after a brain injury; the incidence reaches its highest at about the end of the first year. The fits may be localized (Jacksonian) or generalized, the former usually occurring in the case in which there has been a localized injury to the brain, as happens in a penetrating injury. Epilepsy is more common after severe injuries, and much more common after open than after closed injuries. Its incidence after open injuries, in which extensive scarring may develop in the brain, has been variously estimated as 30 to 50%. Electroencephalography unfortunately does not give reliable information about which individual will and which will not develop epilepsy. In order to diminish this risk it is advisable to prescribe an anticonvulsant to any patient who sustains a head injury in which there has been penetration of the dura mater. Only a small dose (phenobarbitone, ¼ gr. (32 mg.) twice daily) need be taken, but it should be taken for five years without remission. If fits have not occurred at the end of that time it can be discontinued, but withdrawal should be by graduated doses over several weeks. Sudden withdrawal may be promptly followed by a fit.

Next Refresher Course Article.—"The Prevention and Treatment of Tetanus," by Dr. Leslie Cole.

Refresher Course Book.—The first 55 articles of this series (fully revised) have been printed as a book, price 25s. Application for copies should be made to the Publishing Manager, B.M.A. House, Tavistock Square, London, W.C.1.

MEDICAL NEW YEAR HONOURS

The names of the following members of the medical profession were included in the New Year Honours List published in the *London Gazette* on January 1.

Order of Merit

WILDER GRAVES PENFIELD, C.M.G., M.D., D.Sc., Hon.F.R.C.S., F.R.S. Director of the Neurological Institute and Professor of Neurology and Neurosurgery in McGill University, Montreal. Formerly President of the Royal College of Physicians and Surgeons of Canada.

K.C.M.G.

ERIC DENHOLM PRIDIE, C.M.G., D.S.O., O.B.E., M.B., B.S. Chief Medical Officer, Colonial Office.

K.B.E. (Military Division)

FREDERICK HARRIS, C.B., C.B.E., *M.C.*, M.B., B.Ch., D.P.H. Honorary Surgeon to the Queen. Lieutenant-General, late R.A.M.C. Director-General, Army Medical Services.

KENNETH ALEXANDER INGLEBY MACKENZIE, C.B., B.M., B.Ch. Surgeon Vice-Admiral. Medical Director-General of the Navy.

Knighthood

VINCENT ZACHARY COPE, M.D., M.S., F.R.C.S. Consulting Surgeon, St. Mary's Hospital, London.

ARTHUR WILLIAM MICKLE ELLIS, O.B.E., D.M., F.R.C.P. Emeritus Regius Professor of Medicine in the University of Oxford. Consulting Physician, London Hospital.

NORMAN MCALISTER GREGG, *M.C.*, M.B., Ch.M., F.R.A.C.S., D.O.M.S. Honorary Ophthalmic Surgeon to Prince Alfred Hospital and to Royal Alexandra Hospital for Children, Sydney, New South Wales. For services to medical science.

EDMUND BRITTEN JONES, M.B., B.S., F.R.C.P. Honorary Physician, Adelaide Children's Hospital. A leading physician in the State of South Australia. For public services.

PETER MACCALLUM, *M.C.*, M.D., M.Sc., M.R.C.P.Ed., D.P.H. A member of the Medical Board, State of Victoria, and formerly Dean of the Faculty of Medicine and Professor of Pathology in the University of Melbourne.

FRANK PERCIVALE MONTGOMERY, *M.C.*, M.B., B.Ch., F.F.R. Honorary Consulting Radiologist, Down County Infirmary and Down County Mental Hospital. Consulting Radiologist, Ulster Hospital for Children and Women, Belfast. For public services in Northern Ireland.

C.B. (Military Division)

ARTHUR JOSEPH BEVERIDGE, O.B.E., *M.C.*, M.B., B.Ch., M.Sc., D.P.H. Honorary Physician to the Queen. Major-General, late R.A.M.C.

THOMAS NORMAN D'ARCY, C.B.E., L.R.C.P.&S.I., D.O.M.S. Surgeon Rear-Admiral.

EDWARD DEMETRIUS DALZIEL DICKSON, C.B.E., M.D., F.R.C.S.Ed., D.L.O. Honorary Surgeon to the Queen. Air Vice-Marshal, R.A.F.

FRANK KINGSLEY NORRIS, C.B.E., D.S.O., E.D., M.D. Director-General of Medical Services, Australian Military Forces.

C.M.G.

ALFRED EDWARD ROWDEN WHITE, M.D., F.R.A.C.P. A prominent physician in the State of Victoria. For public and philanthropic services.

Hon. C.M.G.

SAMUEL LAYINKA AYODEJI MANUWA, O.B.E., M.D., F.R.C.S.Ed., D.T.M., D.T.H. Inspector-General of Medical Services, Nigeria.

C.B.E. (Civil Division)

IVOR JONES DAVIES, M.D., F.R.C.P. Consulting Physician to the Ministry of Pensions Hospital, Rookwood, Cardiff.

JOHN LOWE, M.D. Senior Specialist (Leprologist), Nigeria.

ROBERT ALEXANDER MCCANCE, M.D., Ph.D., F.R.C.P., F.R.S. Professor of Experimental Medicine in the University of Cambridge. Director, Department of Experimental Medicine of the Medical Research Council at the University of Cambridge.

LOUIS BOYD NEEL, M.R.C.S., L.R.C.P. Conductor of Music.

CHARLES LINDSAY SUTHERLAND, O.B.E., M.D., D.P.H., D.I.H. Senior Medical Officer, Ministry of National Insurance.

ARNOLD LEAROYD WALKER, M.B., B.Chir., F.R.C.S., F.R.C.O.G. Chairman, Central Midwives Board.

ROBERT WEAVER, M.D., D.P.H., F.D.S. Senior Medical Officer, Ministry of Education.

O.B.E. (Military Division)

MORRIS SUTHERLAND WORGAN BISDEE, M.R.C.S., L.R.C.P. Lieutenant-Colonel, R.A.M.C.

O.B.E. (Civil Division)

REGINALD NORRIS O'CONNELL BLANC, M.B., B.Ch. Senior Medical Officer, Tobago, Colonial Medical Service.

ERIC LAUDER CALDWELL-SMITH, M.B., B.Chir. Chief Medical Officer, Shipping Federation, Ltd.

JOHN SINGLETON DARLING, M.B., B.Ch., F.R.C.S. Surgical Specialist, Overseas Food Corporation.

ALFRED CHARLES FISHER, M.D., F.R.C.S. For medical services and welfare work in Northern Rhodesia.

SIMON REGINALD GUNEWARDENE, M.B., B.S. Medical Superintendent, State Home for the Aged, Koggala, Ceylon.

ARTHUR KIDD, M.B., Ch.B. A medical practitioner at Waipiata Sanatorium, Otago, New Zealand.

WILLIAM STEELE MITCHELL, M.R.C.S., L.R.C.P. Acting Government Medical Officer, Grenada, Windward Islands.

WILFRID RAFFLE, M.B., B.Chir. Principal Medical Officer, Ministry of Pensions.

THEODORE HOWARD SOMERVELL, M.B., B.Chir., F.R.C.S. Associate Professor of Surgery at the Christian Medical College, Vellore, Madras.

LEONARD BOOLE STOTT, *M.C.*, M.B., Ch.B., D.P.H. Chief Medical Officer, Papworth Village Settlement.

M.B.E. (Military Division)

PERCY LIONEL GEORGE COLE, L.M.S.S.A. Major, R.A.M.C.

M.B.E. (Civil Division)

LUCIEN NOEL BARTHOLOMEUSZ, F.R.C.S. Visiting Surgeon, General Hospital, Colombo, Ceylon.

JAMES GERRARD CLEARKIN, M.B., B.Ch. Health Officer, West Pahang, Federation of Malaya.

Al-Haj Mohamed PURVIS DRAHAMAN, L.M.S. Medical practitioner. For services to the Malay community in Ceylon.

ERIC PARRY, M.B., Ch.B., F.R.C.S.Ed. Chief Medical Officer to the Kuwait Government.

RICHARD HENRY BARTER SNOW, M.B., B.Chir., D.T.M.&H. Chief Medical Officer to the Bahrain Government.

BERTRAM ALFRED WOOD STONE, B.M., B.Ch. Admiralty Surgeon and Agent, Southsea.

The honour of knighthood is to be conferred also on Arthur Landsborough Thomson, C.B., O.B.E., D.Sc., Second Secretary of the Medical Research Council.

Nova et Vetera

A GREAT PAPAL PHYSICIAN

Giovanni Maria Lancisi (1654–1720) *Aneurysms*. The Latin Text of Rome, 1745. Revised, with Translation and Notes by Wilmer Cave Wright. (Pp. xxxvi+362. $7.50.) New York: The Macmillan Company. 1952.

Giovanni Maria Lancisi (1654–1720) was certainly one of the great physicians, though not among the greatest. He will always be remembered, however, far beyond medical circles as a munificent patron of learning, for he left his wonderful collection of books and manuscripts for public use. The "Lancisian Library" at Rome contains many unique items, and is a resort of scholars. Moreover, Lancisi was responsible for the rescue from probable loss and for the sumptuous publication of at least two scientific documents of the first importance, both by previous papal physicians. One was the collection of anatomical plates of Bartolommeo Eustachio (1520–74)—commemorated in the "Eustachian tubes"—which approach in interest and often surpass in accuracy the figures of Vesalius; the other is the *Metallotheca* of Angela Mercati (1541–93), the foundation-work of the science of mineralogy.

Anatomist and Physician

Lancisi was a native of Rome, where he was educated and medically trained, and where he passed almost all his life. He was a learned, skilful, acute, humane, and extremely successful physician, deeply interested not only in medicine but in science and in the public health. From 1684 he was for 13 years professor of anatomy at the College of the Sapienza, in Rome. He there gained a knowledge of normal bodily structure that served him well in his researches, for he was most zealous in following even his private cases into the post-mortem room. With his young friend Giambattista Morgagni (1682–1771), who—despite the dangers then attending his innumerable necropsies—lived to be 90, he was a founder of morbid anatomy as a science. Many of Lancisi's necropsies are reported with admirable clarity and detail in his *Aneurysms*. They would, in themselves, have justified translation of this long work.

Among other friends of Lancisi were Marcello Malpighi (1628–94), the great microscopist ("Malpighian corpuscles" and "layer"), Bellini (1643–1704) of the "tubules," Pacchioni (1625–1726) of the "corpuscles," and Valsalva (1666–1728) of the "sinuses." In 1688 Lancisi became physician to Pope Innocent XI (1676–89). He soon tired of this court life, and on the Pope's death was glad to return to more general practice. This he never again relinquished, though given various honorific offices by the Vatican and called in consultation by three other pontiffs and many cardinals.

He wrote several works, now classical. Perhaps the best-known are those *On Sudden Deaths* (Rome, 1707) and an account of the outbreak of influenza at Rome in the excessively cold winter of 1709. A physiological curiosity perpetrated by him is that *On the Seat of the Intelligence*—whatever that may mean—which he oddly decided to be the corpus callosum. The raised longitudinal bands on that structure were till recently known as the "nerves of Lancisi." Two of his works entitle him to a place in any account of contagion as a vital phenomenon. One is his *History of the Cattle Plague of 1713 in the Campagna* (Rome, 1715), and the other is *On the Noxious Effluvia of Marshes* (Rome, 1717). In this last he describes five epidemics of malaria and expresses his belief that the disease is conveyed by the bites of insects. The remedy, he rightly decided, was adequate drainage, which he repeatedly urged on the authorities. The work before us was issued posthumously at Rome in 1728, and there are a number of subsequent editions.

Clinical Curiosities

Aneurysms are not nowadays common or often seen except by thoracic specialists. Before the eighteenth century only "external" aneurysms were well known. Particularly familiar were those consequent on careless or incompetent bloodletting at the bend of the elbow. A few "internal" aneurysms had indeed been described by Massa (1498–1569) and Vesalius (1514–64) in Italy, and by Saporta III (1531–73), Baillou (1538–1616), and Du Laurens (1558–1609) in France, but in the seventeenth century they were still regarded as clinical curiosities. The work of Lancisi changed this. Morgagni had studied the older literature and, about 1730, wrote, "There is nothing rare about aneurysms now," for he had in his mind the work of Lancisi. In the eighteenth century "internal" aneurysms seem to have been common in practice and drew much attention, notably from John Hunter.

This impeccable translation is of the high standard expected of the late Mrs. Wilmer Cave Wright. She had long shown her interest in the older medical history, not least in her fine edition of *Fracastor on Contagions* (1930). Our only criticism is that, since the writings of Lancisi were very widely read and are not rare, this edition might have been little more than half the size and price by omission of the very accessible Latin text. Her introduction and notes are as helpful as they are succinct.

It may surprise readers unaccustomed to medical literature of the sixteenth, seventeenth, and eighteenth centuries

that physicians of that period seldom encountered difficulties in obtaining the right to examine the bodies of their patients who had died. Nevertheless detailed records of necropsies before Lancisi and Morgagni are remarkably few. Their scarcity seems due not to any public resistance but to the supineness, distaste, or lack of interest of medical men themselves. The *Sepulchretum* of Théophile Bonet (Geneva, 1679) and its enlargement by J. J. Manget (Geneva, 1700) are supposed to include all known necropsies to date. On these Lancisi and, later, Morgagni had to build.

Space will not permit the quotation of much of Lancisi's book or of Professor Wright's introduction, but an account abstracted by her from one of the works of Cesalpino (1519–1603), another scientific papal physician, is of peculiar interest. It refers to Saint Philippo Neri (1515–95), founder of the Congregation of the Oratory at Rome in 1564. It is probably the most remarkable in any collection of aneurysms. Neri lived to be 80, suffered with violent palpitation of the heart for 50 years, and was canonized in 1622. In 1593 Cesalpino visited the saint, and reported:

"In investigating the cause of the palpitations, I uncovered the chest and found it much wasted, with a tumour near the ribs on the left side, near the heart. . . . At each palpitation the heart rose and fell like bellows."

Twelve days before his death he vomited blood. The day after death a necropsy was held. Two ribs were found detached from the cartilages that normally connect them with the sternum. The heart was much enlarged, the pulmonary artery extremely dilated. When in 1599, in view of the discussion on his canonization, the sepulchre was opened Cesalpino repeated the account of his professional visit to St. Philip.

An Aneurysm Exposed

There are several cases in which Lancisi indicates that one of his aneurysm patients had suffered from syphilis. It is remarkable that he hardly stresses the aetiological association of the two conditions. One case that approaches this problem must suffice:

"A runner aged thirty . . . was no less expert in the pursuit of the goddess Venus than in her gymnastic feats. Though from the latter cause he had often contracted ulcers of the genitals and buboes in the groin, nevertheless, two years before, after becoming much overheated by his accustomed coitus, he felt an annoying and obstinate pain in the region of the kidneys, combined with attacks of erratic fever. This . . . at last increased so much that it was only by bending the trunk of the body that he experienced relief. . . . He entered the hospital but took no remedy except an ounce of cassia, and for three days had no suffering at all except the pain above described. Then, just after he had eaten a dish of food, he breathed his last.

"A death of this kind, unforeseen and unexpected, prompted a close investigation of its causes. We . . . cut open the abdomen, which was full of clotted blood. The viscera, though stained with blood, we found quite unimpaired; we removed them, cleansed them and searched their larger vessels for the opening by which the extravasated blood had flowed out. Whereupon there met our eyes in the trunk of the descending aorta, a little above the emulgent [i.e., the renal] vessels, a remarkably large tumour which, when compressed by an inquisitive hand, discharged a considerable amount of blood like that extravasated; it came from an aperture connected with the abdominal cavity itself."

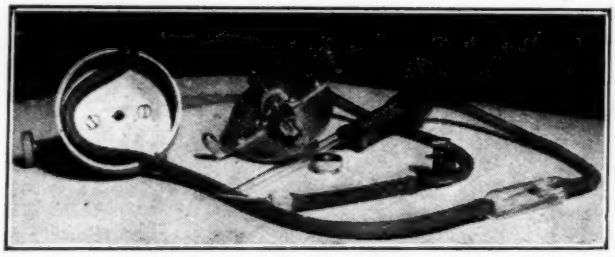
Lancisi, despite his very great success as a fashionable physician, always practised largely among the poor. His real intellectual interest was with science, and he was at least as much an anatomist as a clinician. It is pleasant to recall that this man who moved easily among scholars, magnates, men of science, and princes of the Church left his entire fortune for the alleviation of suffering among the poor, and especially of women of the most needy class.

CHARLES SINGER.

Preparations and Appliances

INFUSION PUMP

Dr. V. KEATING, lecturer in anaesthetics, University College of the West Indies, Jamaica, writes: Below appear some details of a transfusion or infusion pump which has been found useful in the surgical, medical, pharmacy, and pathology departments of a general hospital. It uses a modified form of the "milking tube" principle first described in 1875 by Roussel.[1] The method is simple: a roller, or several rollers, compress a rubber tube against the wall of a circular casing. Moving the rollers by a crank forces fluid along the tube, and no valves or similar moving parts are needed. The contents of the rubber tube do not come in contact with the moving parts of the pump. Machines employing the principle of this instrument have been described by engineers, but have failed to become popular because of mechanical difficulties in design. It is believed that these difficulties have been overcome in the application of the principle to laboratory and medical use. In earlier models a single roller was used, but frothing of blood in the sight-feed of the transfusion apparatus occurred owing to reflux of blood between strokes of the pump, and, moreover, it was not possible to use the instrument for aspiration. Two rollers are employed, therefore, and an adjustment is provided to accommodate any of the sizes of rubber tube



Pump dismounted to show method of inserting the tubing.

normally used in clinical or laboratory practice. The design avoids some difficulties in the models designed by Beck,[2] Riddell,[3] Jouvelet,[4] DeBakey,[5] and others.

An essential feature is that it is not necessary to disconnect an infusion which is already running: it can be speeded up or even reversed without opening the line. To use, a few inches of the infusion line is dropped into the pump casing, the central screw is rotated to suit the diameter of the rubber tube, and the crank is rotated at a speed determined by the patient's needs. The method is thought to be safer, and is certainly more effective, than the use of air pumped into a transfusion bottle. The pressure obtainable is limited only by the strength of the walls of the tubing, so packed red cells or viscous fluids can be pumped through fine needles. It is useful in the pharmacy or laboratory to decant aseptic fluids, and in the anatomy department to inject anatomical specimens. As an anaesthetist I have found it helpful where severe haemorrhage has necessitated rapid blood replacement, and one can comfortably deliver 2 litres in 10 minutes. It has been used on a few occasions as an aspiration pump for septic material.

Made from standard engineering parts, the pump is robust and simple, and occupies little space.

I wish to acknowledge the advice and criticism of the neurosurgical staff of the Military Hospital, Oxford, and the help of Mr. Bax and Mr. Emett, of the Bax Engineering Works, Wheatley, Oxfordshire, who made the instrument.

REFERENCES

1 *Arch. gén. Méd.*, 1875, **25**, 129, 443, 690.
2 *Ergebn. inn. Med. Kinderheilk.*, 1926, **30**, 150.
3 *British Medical Journal*, 1939, **1**, 1125.
4 *Bull. Soc. méd. Hôp. Paris*, 1934, **50**, 537.
5 *New Orleans med. surg. J.*, 1934, **87**, 386.

Correspondence

Because of the present high cost of producing the Journal, and the great pressure on our space, correspondents are asked to keep their letters short.

Pulmonary Moniliasis

SIR,—I foresee disappointment if readers of the *Journal* are tempted by the unusual experience of Drs. Osman Barlas and Muzaffer Akyel (December 27, p. 1394) to treat pulmonary or any other form of moniliasis with penicillin and aureomycin. The growth of *Candida albicans* is entirely unaffected by either of these antibiotics, except in so far as it is actually stimulated. Moniliasis, oral, intestinal, and pulmonary, is a well-recognized complication of therapy with aureomycin and other newer antibiotics, of which repeated warnings have been given: minor forms of it are common and fatal ones have been reported.

Any alternative explanation of the response to antibiotic treatment in this case can only be tentative, but I venture to point out (1) that no mention is made of the bacterial flora of the sputum; (2) that Fig. 3, illustrating *C. albicans* in the sputum, also shows bodies which resemble cocci, one pair even appearing faintly capsulated; (3) that if the lesion was a lung abscess due primarily to a pneumococcus or staphylococcus the favourable response is not so extraordinary. The presence of *C. albicans* in the sputum could have been due to an associated bronchial moniliasis, a much commoner and less serious form of this infection than one involving the lung parenchyma. I assume from the description of the repeated finding of the organism in direct films that it was in the bronchial secretion itself and was not a contaminant from the mouth. It exists in the majority of mouths, and the interpretation of its presence in sputum always calls for caution.—I am, etc.,

Harpenden. LAWRENCE P. GARROD.

The Neurology of John Hunter's Last Illness

SIR,—While reading Sir Russell Brain's interesting and illuminating article (December 27, p. 1371) on "The Neurology of John Hunter's Last Illness" my memory recalled the early years of the 1880s when I had the privilege of working with Sir Hughlings Jackson at the London Hospital as his clinical clerk, house-physician, and for a short time afterwards.

I remember his many bedside addresses at which he often used to say, regarding nerve diseases, that when a case presents a medley or mixture of symptoms which do not appear to communicate with each other, when there is an incoherent succession of symptoms, or when there is a random association of symptoms, *strongly suspect syphilis*, although there be no actual demonstration or history thereof. This was long before the introduction of the scientific methods available now.

Sir Russell Brain's narrative is a good illustration of the profundity of Sir Hughlings Jackson's observations so many years ago.—I am, etc.,

Manchester. WILLIAM COATES.

Modified P.A.S. for Treatment of Genito-urinary Tuberculosis

SIR,—Ever since P.A.S. has been used in the treatment of tuberculosis, its usefulness and value have been materially reduced by the considerable degree of subjective toxicity which is encountered in many of the patients. Although admittedly rather mild in the majority of cases, these toxic symptoms have nevertheless been severe enough in a number of cases to necessitate cessation of treatment, at least for a time. Our experience in treating cases of genito-urinary tuberculosis has proved to be no exception, and we have found slight toxicity, mainly nausea, vomiting, and anorexia, in about 65% of cases, and severe toxicity in 40%. As might be expected, we found that this toxicity was worst with higher doses, and also increased with increased duration of treatment. Eventually the problem became sufficiently serious to make us consider limiting treatment to a period of one month followed by one month's rest, and this was put into effect some time ago.

A modified form of P.A.S.—namely, the sodium salt of N. benzoyl P.A.S.—then became available, and Debré[1] demonstrated its effect in the treatment of serious cases of genito-urinary tuberculosis. This drug, while having many advantages over P.A.S., has the disadvantage of being extremely unpleasant to take, necessitating administration in the form of cachets. Quite recently we have been able to obtain supplies of the calcium salt of N. benzoyl P.A.S. (calcium B.P.A.S.), which proves to be almost completely tasteless.

Briefly, the advantages of this drug are as follows: First and most important, it is completely non-toxic, both subjectively and objectively, and it has been found to have none of the unpleasant sequelae of P.A.S. We have been using this drug now for over a year, and have treated about 80 cases. All the patients have been given this drug continuously (15 g. daily for six months), and in no case have we had to decrease the dose or stop the drug. None of the patients has complained at any time of its effect. Secondly, it is completely tasteless. Thirdly, it need not be administered so frequently as P.A.S., since a fairly constant P.A.S. blood level is obtained, and we normally give the drug at intervals of eight hours. After a single dose of 8 g. of calcium B.P.A.S., P.A.S. itself can be detected in the urine up to 36 hours later. From our experience with this drug, we feel that the above daily dose of 15 g. is perhaps too low, and we are now advocating 7 g. at intervals of eight hours. Using this dose, specimens of urine tested every two hours during a 24-hour period showed a maximum concentration of 85 mg.% of P.A.S. and a minimum concentration of 30 mg.% of P.A.S. It is this reasonably constant urinary concentration of P.A.S. which has proved so advantageous in genito-urinary tuberculosis.

One of the most valuable properties of P.A.S. is that it prevents the emergence of strains of tubercle bacilli resistant to streptomycin. Calcium B.P.A.S. seems to have retained this property, as we have encountered only one new case of streptomycin resistance in 80 cases of genito-urinary tuberculosis treated for six months.

Calcium B.P.A.S. appears to be relatively inactive against tubercle *in vitro*, and this suggests that its effect depends upon the production of P.A.S. in the body. It seems likely that the relatively insoluble calcium B.P.A.S. is first broken down to free B.P.A.S. presumably in the gastro-intestinal tract, and this B.P.A.S., after absorption, is hydrolysed to produce free P.A.S. in the blood. The activity of calcium B.P.A.S. would therefore seem to rely upon the level of free P.A.S. produced by its breakdown. It is hoped to publish a full report on this work at a later date.

I should like to thank Herts Pharmaceuticals, Ltd., for supplies of calcium B.P.A.S., and also for their encouragement and valuable help.—I am, etc.,

Liverpool. J. G. Gow.

REFERENCE

[1] *Presse méd.*, 1950, **58**, 647.

Intra-arterial Injection of Tubocurarine and Thiopentone

SIR,—A married woman aged 67 was brought to the theatre to undergo radical resection of malignant glands secondary to carcinoma of the vulva. A Gordh needle was inserted into a vessel overlying the dorsi-lateral aspect of the lower end of the right radius, which was taken to be the cephalic vein, and a test dose of 5 mg. of tubocurarine chloride ("tubarine"), non-miscible, injected. The patient immediately stated that she experienced a burning pain in the forearm, and a few seconds later that the pain had disappeared. A further dose of 10 mg. of tubocurarine chloride was then injected without any unusual effect, and a slow injection of 2½% solution of thiopentone begun. Pain was felt immediately both in the hand and forearm, and so the injection was stopped after 1 ml. had been given, and the top of the needle unscrewed. It was evident now that the needle was in an artery, as the escaping blood pulsated. Thiopentone, 350 mg., was immediately injected into a vein in the other arm; the patient was intubated without difficulty, and aided respiration was started with an equal mixture of nitrous oxide and oxygen. Meanwhile 10 ml.

of normal saline was injected through the Gordh needle, followed by 2 ml. of 4% procaine, and 15 minutes later both these injections were repeated.

The immediate effects of the intra-arterial injection were a reddening of the skin over the lateral aspect of the forearm with urticarial appearance after a few minutes, and oedema of the thumb and thenar eminence without any apparent effect on the skin circulation of this area. The radial and ulnar pulses remained normal throughout the episode. The skin effect was markedly lessened after the first injections of saline and procaine, and disappeared entirely after the second, while the oedema of the thumb was much reduced by the end of the operation—which was completed without incident in one and three-quarter hours—and had disappeared three hours later. There were no detectable ill-effects the next day or subsequently.

The interest of this case may be summarized thus: (1) It must be most unusual to encounter an artery of sufficient size to accommodate a Gordh needle at this site in the presence of a normally situated radial artery. (2) The tubocurarine chloride, though plainly irritant, must have passed through the capillary bed and reached the general circulation, because the patient was well relaxed when intubated. (3) The thiopentone appeared to produce vasodilatation instead of the more usual spasm. This was most strikingly shown by the immediate blanching of the erythema when the blood was displaced by the saline injections. This, I think, was probably due to the weakness of the solution and the small amount of thiopentone injected. (4) The case illustrates the importance of leaving the needle in the vessel rather than withdrawing it at once, as recommended by some authorities. Also it is advisable to allow blood to flow through the needle while the saline and procaine injections are being prepared, because if a clot forms there is no way of dislodging it without a serious risk of blocking an artery further on. This might result at least in a troublesome skin ulcer, and it would be very difficult to find such a small vessel again with a fresh needle.

I am indebted to Mr. K. M. Mackenzie for permission to publish this case.—I am, etc.,

Colchester. J. N. FELL.

The Backward Child

SIR,—*Multa sed non multum* is a criticism that might be levelled against "The Backward Child" in the Refresher Course for General Practitioners (December 13, 1952, p. 1304) by those who wish for simple but accurate information on specialties. Inevitably, the crowding in this article has produced misleading statements which urgently require comment. May I select two?

The article says, "There is no general agreement on the nature of intelligence, and tests . . . the Binet-Simon . . . the Vineland scale often give very different results in the same patient." While the first part of the statement is correct, this should not be taken to mean that the various opinions on the nature of intelligence lack precision, and much less even that validity, reliability, etc., of tests are undefined. It certainly is practically difficult, though not impossible, to assess accurately the I.Q. of the pre-school child, nor is this clinically imperative for the reasons implied, as "certification" of educationally subnormal children does not happen at this age, and global developmental assessment is both more revealing and accurate. Affective factors will be recognized by the expert. But, what is more serious, surely Dr. Kirman knows that the Binet-Simon (its current revised Terman-Merrill revision) is neither "educational," especially not at the pre-school level, nor does it purport to measure the same as the Vineland Scale of Social Maturity. For the older child, other tests well differentiating "efficiency" and "potentiality" of intellect are available—for example, Wechsler's Bellevue extension for children. Intelligence testing is, in the hands of the expert, both valid and reliable. There are, of course, well-recognized limitations. For a general account see my "Intelligence and the Limitations of Testing" (*Schoolmaster*, June 9, 1949).

Dr. Kirman says, "There are . . . many backward children, perhaps the majority, in whom the defect is functional and perhaps only temporary." This is quite true, if backward means backward in school subjects. This backwardness can be assessed psychometrically, and treated educationally and psychotherapeutically. See Schonell, F. J., *Backwardness in the Basic Subjects* (Edinburgh, 1948), and my own "Psychotherapy of Children Jointly with their Parents" (*Zeitschrift für Kinderpsychiatrie*, 1951, **18**, 33). If Dr. Kirman refers, as he should according to his topic, to the pre-school child, he must mean intellectual and/or emotional backwardness. The former is accurately assessable with the aid of developmental tests—for example, Gesell's—by the trained educational psychologist and child psychiatrist, with high reliability, the latter by observation and interview, including history, the validity and reliability varying, naturally, with the interviewer's insight and experience. Leaving "moral" certifications aside, no child need run a risk of certification on evidence—or lack of it—as indicated in the article. Certainly no child psychologist or psychiatrist would be deceived by the mentioned "shyness" or inhibited co-operation of the child, as alleged.

For fear of intolerably trespassing on your space, I shall limit myself to the above remarks.—I am, etc.,

Sedgefield, Co. Durham. D. J. SALFIELD.

Abolition of Flogging

SIR,—In a book review (December 20, 1952, p. 1343) Dr. E. B. Strauss writes: "However, the present moment (when for example so many otherwise responsible people are shrilly and irrationally clamouring for the reintroduction of flogging) cannot, alas, be regarded as propitious for the dispassionate review of the laws . . ."

Whether a reviewer should air his fads, even in brackets, when they have nothing to do with the book is doubtful, but if he does he is liable to be challenged. I and many others are appalled at the huge sentences—10 years on a youth of 18, 12 years on one of 20—that the judges have found it necessary to pass because flogging has been abolished. Is it irrational and irresponsible to agree with the Lord Chief Justice in deploring this necessity and advocating a return to old methods? Has Dr. Strauss considered what would be his own present state if he had been from the age of 18 to 25 in jail? His use of the word "shrilly" shows that he is trying to prejudice the reasoned case for a return to flogging, which in my view would be a retrograde step made necessary to meet a retrograde state of affairs, and, compared with its alternatives, a merciful one.

One finds these jeers from psychiatrists particularly irritating, because it was their paralysing fairness, their pampering unreality, their dislike of discipline, their airy theorizing and failure to foresee the results of their own teaching that contributed not a little both to the abolition of flogging and to the volume of violent crime which has made its return an apt necessity.—I am, etc.,

Rye. C. G. LEAROYD.

Specialists in Physical Medicine

SIR,—There are many reasons why the proposed increase in the number of consultants in physical medicine outside teaching centres should be resisted. It is true that clinicians have but a poor understanding of the value and limitations of physical medicine techniques, but this is more than outweighed by the necessarily limited knowledge possessed by specialists in physical medicine of clinical medicine and surgery, fields from which their patients are drawn. Unfortunately the ignorance of clinicians will not be cured by the appointment of more consultants in the specialty. A sounder approach would be better practical teaching and demonstration of the subject in the medical schools.

Personal experience of working in hospitals where cases requiring physiotherapy were first referred to a specialist in the department soon revealed the disadvantages of the system. Such patients were frequently "lost" for months, or became "interesting cases," only to return with a more flagrant neurosis than when they were referred for three weeks' active exercises. Very often the recommended treatment was not given, but something more exotic substituted. Treatment of patients with neurotic potentialities in such a

way can be harmful and wasteful, and all too frequently the patient's "back" or "shoulder" is treated as something quite detached. Physiotherapy in various forms can be most valuable in the management of psychoneurotic, psychosomatic, and somatopsychic states, but it requires careful handling. At present too many of these patients are referred for physiotherapy as a gesture of despair, and the department becomes overloaded and in some instances seriously abused by clinicians. It should be noted that the abuses of *x*-ray and pathology departments, though well supplied with directors, cannot be prevented.

It can be said that all enthusiasms in medicine find a level in time, and it is very possible that some new development in the rheumatic field will leave the physiotherapy departments relatively empty. It is certain that further splintering of clinical responsibility in the way suggested can only react to the patient's detriment. If it is a crude attempt to impose a control, there have already been enough examples to show that it is likely to be a sterile, costly, and inefficient method of doing it.—I am, etc.,

Ipswich. J. W. PAULLEY.

Iron Therapy

SIR,—The article by Dr. D. Haler (December 6, 1952, p. 1241) indicates that ferrous gluconate given with a mixture of vitamins is a more efficient haematinic than ferrous sulphate or iron ammonium citrate given without those vitamins. This leaves us quite in the dark as to whether the vitamins play any part, and if so what part, in the process. Possibly they only act as a reducing agent or in some other non-specific way.

Surely what is required is a comparison of the effects of equivalent quantities of the iron compounds alone, and then, if indicated from previous investigations, with the same added vitamins or reducing agents, etc.—I am, etc.,

Brighton. R. S. SAXTON.

Christmas Disease

SIR,—Doubtless several thousand readers, like myself, eagerly unrolling your Christmas number (December 27, 1952) and hastening through its pages to discover what seasonable though morbid experiences had been enjoyed by no less than seven distinguished haematologists (p. 1378), were similarly disappointed to find that this festal title was merely a ferial eponym for an all-the-year-round disease, derived from the name of one of its victims.

Now, Sir, eponymous names for diseases are disagreeable things and dangerous to handle. It is true that they offend least when they originate accidentally, as here, from the name of the first recognized sufferer; such an eponym, chosen undesignedly, is devoid of aetiological, nosological, proprietary, or historical significance, if it is chosen with discrimination. But to use the name Christmas shows that the seven authors lack perspicacity, for here is a word universally known in one meaning which they propose to perpetuate in another and excessively uncommon usage, that of a proper surname. The object of the surnominal eponym is thereby defeated, since it cannot be recognized as such without an explanation. Could not the authors foresee how tedious, and indeed irritating, it will be to all teachers to have to refer always to "Christmas disease—it has no connexion with December 25; Christmas happened to be the name of one of the first patients recorded by Biggs *et al.* (1952)"? Confusion is not, alas, a matter of semantics alone. It appears that the "Christmas factor" is something that one has when *not* suffering from "Christmas disease." The "Christmas factor," it is related, is something that Master Christmas, aged 5, had not, and, lacking it, he manifested "Christmas disease." We do not yet refer to scurvy as "vitamin-C disease," and even the more fanciful "L-E factor" is something which a patient with lupus erythematosus is reputed to have in a positive way.

The communication published under this ill-considered title is emphatically one of the greatest interest and importance, for it reveals the nature of one variety of bleeders' disease, the existence of which is, as the authors say, of great academic interest, while its recognition in a specific patient may be of vital importance. The matters at which my raillery is aimed may be irrelevant to the intrinsic value of the research, but they are matters so open to mockery that they may well prove disadvantageous to its proper esteem.

Some parents live to regret the names they have thoughtlessly bestowed upon their innocent children. Would it not be possible for at least a quorum of the progenitors of this disease, in collaboration with you, Sir, in your capacity as registrar of its birth, to substitute some less ridiculous and more appropriate name?—I am, etc.,

Leeds. DOUGLAS H. COLLINS.

SIR,—While congratulating the authors of the paper on "Christmas Disease" (December 27, 1952, p. 1378), it is perhaps permissible to wonder whether "the naming of clinical disorders after patients" is altogether appropriate in this instance. The word "Christmas" usually suggests the great Christian festival rather than an individual's name, so that we cannot complain justifiably that we first read about the new disease in a "topical" paragraph in the lay press on December 24. Case 2 might have been chosen for purposes of nomenclature; we can be reasonably confident, I suppose, that his name was not Easter?

Fowler would solve the difficulty for us no doubt by a reminder that usage dictates an apostrophe—"Christmas's disease."—I am, etc.,

Birmingham. P. R. KEMP.

The College of General Practitioners

SIR,—I wonder if you will permit one criticism of the report of the General Practice Steering Committee published in your issue of December 20, 1952, p. 1321? After an excellent survey of the whole project, the report gives three alternative qualifications for foundation membership of the college, and I would like to comment upon each.

Twenty years in general practice may, indeed, give that wealth of experience which should qualify a man for foundation membership, but this is surely a dangerous criterion, as it is in general practice that there exist the most favourable opportunities for stagnation and rapid deterioration to be found in the whole of medicine. The second qualification is essentially sound, in that it requires a doctor to make an effort to keep up to date. It is interesting to speculate what might be required of this doctor when he attains to his 20 years' practice. Is he then "written off," and allowed to stagnate? The third is the most dangerous of all. The possession of a higher degree or diploma by a general practitioner may well imply that he has embarked upon a specialist career, only to fail and turn embittered and dissatisfied to general practice. As such, and quite apart from the question of owing allegiance to any other college or faculty, is he not just as likely to stagnate as the man who has set out from the first as a G.P.?

There is no doubt in my mind that there can be but one sound qualification for foundation membership. This must require (*a*) a modicum of experience in general practice, and (*b*) an undertaking to keep up to date in some way *until death or retirement.* Given this, any other criterion becomes superfluous.—I am, etc.,

Upton, Hunts. JOHN K. PATERSON.

SIR,—The report of the General Practice Steering Committee (December 20, p. 1321) requires vigorous and widespread opposition. Some of its aims are praiseworthy and, it is to be hoped, will soon be fulfilled; but laudable aims are always essential to the acceptance of such a report. A better title would be "The Board of Control of General Practice."

"The College will give a lead to general practice."—We have not asked to be led. We don't even want to be steered, and we need leaders less than we need a large

body of doctors who are persistent in helping to improve the medical service. "The College will raise the status and enhance the prestige of general practitioners."—Status and prestige are part of the stock-in-trade of the witch-doctor and medicine-man, but they come naturally and uninvited to the conscientious worker and the skilled craftsman, who do not need to subscribe to have their status raised or their prestige enhanced. It would be better if we ourselves avoided the ten-guinea method. Surely we do not feel the need to pay for our elevation by self-appointed leaders of the profession. "Postgraduate teaching."—I hope that I am deriving some benefit from occasional attendance at ward rounds, clinics, and lectures by more experienced practitioners, but I refuse to be directed to any compulsory lectures by the appropriate subcommittee of the College of General Practitioners.

"Principals may be helped in finding suitable assistants."—Principals need no such help. There are usually more than fifty applicants for every assistantship with view to partnership. We would be better occupied in helping assistants to become principals. "Criteria for Foundation Members . . . How will practitioners be able to join at first?"—The "at first" sounds ominous—it suggests more subtle methods to come to restrict entry into the high-prestige group. I foresee the membership of the College of General Practitioners being used as a method of further restricting entry into general practice.

The date of starting of this new college was January 1. It would be far less sinister had January 1, 1954, been arranged, but this college which it is hoped to thrust upon us depends for its formation on an administration being set up speedily with no better method of opposition than a few pathetic letters to the medical journals. No doubt it is hoped that foundation members will clamour to join so as not to miss the bus. It would have been more thorough to have issued an edict that the closing date for application would be December 31, 1952. The National Health Service is obviously continued at the wish of the majority of the population. Of this college it is stated, "The leadership must be truly national." This is a meaningless slogan. It is essential that control of such a college must lie with elected representatives (not leaders) under a proper constitution with periods of office restricted by time limits. The report of the Steering Committee says, "We realize that final details of the College must be decided by general practitioners themselves." This sounds well, but the real issue is, How are these decisions to be made by over 20,000 G.P.s? Perhaps by self-appointed spokesmen. In your leading article (December 20, 1952, p. 1344) you say, "That some kind of distinction be sought is inevitable." Undoubtedly this is a shrewd forecast.

We must be optimistic and trust that there will be a counterblast, a loud outcry from G.P.s which will cause an abrupt halt to these edicts, and that there will be an incessant demand for less leadership from above, and every opportunity for full discussion, criticism, and control of any schemes which come from status-raisers, steerers, leaders, prestige-enhancers, and entry-restricters. If this is not forthcoming we are doomed to a sheep-like mediocrity.—I am, etc.,

Marple Bridge, Cheshire. BERNARD KENDRICK.

SIR,—In your leading article on the College of General Practitioners (December 20, 1952, p. 1344) you express disappointment that the college may in time decide to issue a diploma and at the same time suggest that elevation to the fellowship of the college might be by election, as is the custom of the Royal College of Physicians. The Royal Colleges of Physicians conduct membership examinations and the diploma is recognized as an essential to the consultant physician. It is to be hoped that a similar test of eligibility for membership of the College of General Practitioners will be introduced at an early date.

There is an urgent need in this country for improvement in prescribing, which is no longer dependent on a knowledge of the *B.P.* or *B.P.C.* but is largely decided by the tidal wave of advertisements for proprietary preparations which deluges the profession. I am not referring to the scientific preparations which have, as the result of patient research, proved to be of inestimable value to our patients. The membership examinations which I hope the College of General Practitioners will decide to conduct should in my opinion demand, along with other subjects, an up-to-date knowledge of therapeutics and *materia medica*. If the College of General Practitioners is to win the respect of the medical profession throughout the world and eventually become worthy of a Royal Charter, membership of the college should be given only to those who have on their own showing proved themselves worthy of the honour.

The one part of the report with which I find myself at variance is the decision to offer foundation membership to practitioners of five years' standing who promise to accept postgraduate instruction for three days each year. One realizes that entry on the conditions stated applies only to foundation members, but I venture to suggest that membership of the college at its inception should not be too freely granted. At the present time there is no diploma in general medicine to which the young practitioner may aspire while carrying out his work as a family doctor. The M.C.G.P. might well become the hallmark of a first-class general practitioner, but this desirable state of affairs is not likely to arise if entrance to the college as a member can be obtained by presenting a cheque for £10.

Apart from the above criticism, I have nothing but praise for the report of the General Practice Steering Committee. The members of the committee are to be congratulated on their courage and vision in establishing a College of General Practitioners. I hope to become a member of the college, but would be content to be an associate until the Foundation Council has drafted provisional by-laws.—I am, etc.,

Edinburgh. ALISTAIR G. CRUIKSHANK.

Advertising in Examinations?

SIR,—Drs. H. C. Churchill-Davidson and W. D. Wylie (December 20, 1952, p. 1361) draw attention to the use of trade names of drugs in the examination for the Diploma of Anaesthetics. Unless anaesthetists are very different from their colleagues in hospitals, this is probably necessary if the candidates are to know what drug is being referred to. I recently asked a hospital physician what dosage of sulphadimidine he advised. He said he could not help me as he always used "sulphamezathine." It would not surprise me to find that an anaesthetist denied knowledge of, for instance, thiopentone.

Without any prolonged research, I find that I have received letters from hospital doctors in which the following substances are referred to by trade names: acetarsol, amphetamine, butobarbitone, chloramphenicol, icthammol, isoprenaline, menaphthone, and phenobarbitone. This is not entirely a matter of pedantry, for manufacturers often charge more for a drug under a trade name than under its official name. The Ministry of Health has pointed out to general practitioners that £242,000 could have been saved if dexamphetamine had been prescribed in 1951 instead of "dexedrine." I hope the Ministry will also address a few words to hospital doctors.—I am, etc.,

Orpington, Kent. L. M. FRANKLIN.

Anaphylaxis after Penicillin

SIR,—The following account of anaphylaxis after penicillin may be of interest and use to other doctors. A man aged 63 has had thrombophlebitis migrans (without any evidence of neoplasm in his intestinal tract) for the past three years. On many occasions I have given him "distaquaine" suspension of penicillin G, always in doses of a mega unit a day for four or five days. About a month ago I gave him such a course, and after the last injection he complained of a little drowsiness and faint tingling in his hands. A week ago he began another patch of thrombophlebitis. He had a tender, swollen, and inflamed hard vein in his leg, with a slightly raised pulse rate and a temperature of 99° F. (37.2° C.). I gave him a mega unit of distaquaine penicillin, and within two minutes he complained of feeling drowsy; then his face went red and he began to belch; in another few seconds he became unconscious with stertorous breathing; his face became pale and cyanosed, his pupils dilated, and his pulse just perceptible at the wrist. He became incontinent of urine and faeces, and appeared to be going to die. I gave him 10 minims (0.6 ml.) of 1:1,000 adrenaline, and within a minute he began to recover. He started to

sweat and breathe regularly, his pulse returned, and his pupils contracted to normal size. Within five minutes he was able to help himself into his bed.

The lessons to be learned from this case are that the patient's description of symptoms should always be listened to and given due weight, and that some adrenaline should always be carried in the bag alongside the penicillin.—I am, etc.,

Solihull, Birmingham. ARTHUR BEAUCHAMP.

Self-demand Feeding

SIR,—I should like to support Professor R. S. Illingworth in his criticisms (December 20, 1952, p. 1355) of the comments made by Drs. J. D. Hay, P. D. Moss, and M. M. P. Ryan (November 29, 1952, p. 1180) about Professor Illingworth's earlier paper on self-demand feeding.[1] For many years now the profession has been teaching, through the agency of university departments, maternity units, infant welfare centres, and textbooks, a rigid system of infant feeding. The kinds and quantities of food, the time of feeding, the length of the feed and so on, have been insisted upon in such a way that mothers are now merely the agents and nursemaids of complacent authorities.

For far too long we have blamed the mothers when things have gone wrong, and there has even developed a curious belief that mothers can have a bad psychological effect over their babies. In maternity units babies are still kept segregated together in nurseries when they should be at the mother's bedside, thereby providing a natural psychological influence, and when they leave the maternity unit we have imposed on the mothers rules and regulations which have not infrequently led to the very troubles for which we have blamed the mothers. These rules and regulations have been laid down by all kinds of odd people who have been accepted at the time as authorities on the subject, and many of us have sheepishly continued to teach and practise them. Who, for instance, invented the four-hourly feed for the small infant? Who advised the quite inadequate feeding quantities for most babies on the packets of dried milk, and who laid it down that the infant should take 20 minutes over a feed and should be fed with a bottle open at one end only, which means that the infant is always pulling against a partial vacuum? Many so-called feeding disorders are due to wrong technique and underfeeding in quantity and/or quality of food, and some teaching units still continue to teach that overfeeding is a danger and that healthy babies suffer from fat intolerance and require skimmed milk during the early weeks and months of life. Those who instruct sister tutors, nurses, midwives, and medical students have a heavy responsibility, and it is for this reason that I strongly support Professor Illingworth in his endeavour to break away from these rigid feeding schedules and allow mothers to take a more natural and common-sense attitude towards their babies.

May I therefore at this season of the year join with Professor Illingworth and Dr. B. S. B. Wood,[2] who has recently shown that vomiting in infancy is not infrequently due to underfeeding, and with Dr. I. G. Wickes,[3] who has challenged the idea that overfeeding is a common event, in wishing all those babies to be born in 1953 a less "windy" and more bountiful and natural New Year.—I am, etc.,

Leeds. WILFRED VINING.

REFERENCES

1 *Lancet*, 1952, 1, 683.
2 Ibid., 1952, 1, 28
3 *British Medical Journal*, 1952, 2, 1178.

Period of Gestation

SIR,—Recent letters suggest theories on inherited variations in the duration of gestation and revive the worn-out idea that the length of gestation is 10 times the menstrual interval. I hope I may be forgiven if I point out that these valuable letters pay too little attention to facts which are accepted by workers who have devoted themselves to the study of menstruation, ovulation, and fertility.

First, although a woman's menstrual cycles may seem to be regular in time, they are really not. Holt[1] described the woman whose periods recur regularly as a *Märchenprinzessin*—a princess out of a fairy story. I took part in some accurate recording of menstrual cycles of normal women (Gunn, Jenkin, and Gunn[2]) in which 479 women participated for at least a year, and we found that the woman with the least variation of all had 2¼ days' difference between the shortest and longest cycles. This was exceptional, for in 84% there was a difference of six days *or more* between the longest menstrual cycle and the shortest. Allen,[3] Latz,[4] and all who have obtained accurate records agree with this.

Secondly, the experts do accept that ovulation is related by a constant time interval to the following menstruation and not to the preceding one. Usually, but not invariably, it is 16–12 days before the next menstrual period begins (Hartmann[6]; Krauss[8]; Venning and Brown[7]; Rock[8]; Palmer[9]; and Bowes[10]).

Thirdly, it is well known to gynaecologists that the duration of the menstrual cycle shortens as a woman gets older. In our study of normal women published in 1937 we were able to show that on an average the cycle gets shorter at the rate of one day every five or six years. Thus in this country the average length cycle for all women is 29 days, but for the 15-year-olds it is 32 days and for the 45-year-olds it is 26 days.

As a working rule we accept the period of gestation dating from the first day of the last menstrual period as nine calendar months, plus seven days or 40 weeks or 280 days (conventional). As the average woman with a 29-day cycle ovulates 16–12 days before the expected onset of her next menstrual period, the duration of pregnancy presumably averages 265 days (actual). I find this knowledge useful when I am faced with a suspected case of post-maturity, and one of the questions I ask is what is the duration of the menstrual cycle. Sometimes I find that it is six or seven weeks, and I realize that ovulation and fertilization may have taken place two or three weeks later than in the average case so that post-maturity is not proved and is in fact unlikely. The idea that the period of gestation may be 10 times the average menstrual cycle is repeatedly postulated. If this were true it would mean that the old would have very much shorter pregnancies than the young, and the average woman of 45 would have a pregnancy of 260 days, compared with one of 320 days at 15 years of age.

Dr. Joyce Mulvany and Mr. Goldstone (November 15, 1952, p. 1102) evidently have children with a gestation period about 13 days shorter than the average, and the short pregnancy seems to be habitual. I am convinced from many years' observation that every woman has a characteristic gestation period which tends to recur in all her pregnancies, some going into labour at the expected date, some a week or 10 days early, and some consistently late, but in each one there is a pattern which is only altered by some exceptional factor. It would indeed be worth while, as the writers suggest, to investigate whether this characteristic gestation period is inherited and whether the paternal inheritance in the foetus plays any part. It is a matter for faithful case recording of large numbers of patients whose histories may still be available when the next generation is producing children.—I am, etc.,

London, W.1. ALISTAIR GUNN.

REFERENCES

1 *Marriage and Periodic Abstinence*, 1937, 1. London and New York.
2 *J. Obstet. Gynaec. Brit. Emp.*, 1937, 44, 839.
3 *Amer. J. Obstet. Gynec.*, 1933, 25, 705.
4 *J. Amer. med. Ass.*, 1935, 105, 1241.
5 *Contr. Embryol. Carneg. Instn*, 23, 1.
6 *Periodic Fertility and Sterility in Women*, 1934, trans. D. H. and K. Kitchen. Vienna.
7 *Endocrinology*, 1937, 21, 711.
8 *New Engl. J. Med.*, 1941, 225, 910.
9 *Rapp. 10me Congr. franc. de Gynec.*, 1946, 10, 69.
10 *Modern Trends in Obstetrics and Gynaecology*, 1950, 524. Butterworth. London.

The Catheter and the Prostate

SIR,—Mr. Stephen Power (December 6, 1952, p. 1256) would have us go back to the old septic days of prostatectomy because he now has streptomycin to remove—or not to remove—the infection that he has caused. "Aseptic prostatectomy" and the invariable one-stage operation with immediate closure of the bladder was practised five years before any antibiotic was available in this country. Asepsis with the urethral tube passed from the bladder outwards and left in for two (not five) days or less will result in the patients leaving the hospital in 10 days with sterile urine in four out of five cases. It has reduced an appalling mortality to 2% (see *Prostatectomy*, by Professor

Charles Wells). Has Mr. Power really ever removed a prostate with the asepsis of a knee-joint surgeon? The proof of an aseptic prostatectomy is this. In over 200 herniotomies, appendicectomies, radical cures for hydrocele, etc., performed *after* the prostatectomy, at the same sitting and with the same instruments, not one has suppurated.

Prostatectomies for acute retention at midnight, when theatre asepsis is at its lowest, are unnecessary: the suprapubic drainage needle will suffice until morning—or until the patient arrives from overseas. Delay for full investigation is always advisable when the diagnosis, or necessity for operation, is uncertain. The catheter passed "with the full ritual of asepsis in a well-equipped hospital ward" (a room now full of antibiotic-resistant strains of bacteria) is more dangerous than one passed with proper care in the meanest cottage of this country. To the surgeon a preliminary cystostomy is a confession of defeat, of an incomplete asepsis or haemostasis; to the remote practitioner who has to pass a catheter I give every sympathy.

The atonic bladder, I agree, will always be a problem; but it is easier to eliminate sepsis in the absence of obstruction. This is a cardinal rule of surgery. Mr. Power really cannot render aseptic the cesspool of the post-prostatic pouch by tapping the clear urine above by a catheter or drainage uphill through a cystostomy tube.—I am, etc.,

Manchester. WILSON HEY.

SIR,—Although he makes no direct reference to my recent monograph[1] on this subject, Mr. Stephen Power's verbatim quotation from it in his letter (December 6, 1952, p. 1256) obliges me to reply to him. In effect, what Mr. Power says is that he disagrees. So he may—I take no exception to that. He then asks why cases of chronic retention should not be drained by catheter until fit for surgery. This I mind very much. The whole of my short book is devoted to developing the answer to precisely this question. Even with strict economy in the use of words, it takes 80 pages of closely reasoned argument. It would be futile to attempt to cover the same ground in a short letter, and I do not intend to try the impossible.

In my opinion the principles advocated by Mr. Wilson Hey can be applied with great benefit to the patient. Mr. Hey has already with remarkable patience borne the brunt of the sort of criticism always levelled at a pioneer. Recently his views have gained wide acceptance. They deserve close study, and I do earnestly suggest to Mr. Power that, though I may have failed to convince him, I have made a serious attempt to answer in my book the very questions which he asks.—I am, etc.,

Liverpool. CHARLES WELLS.

REFERENCE

[1] *Prostatectomy—a Method and its Management*, 1952. Livingstone.

Mind and the Skin

SIR,—Dr. F. L. Lydon's letter (December 20, 1952, p. 1356) under the heading "Mind and the Skin" opens a larger question. It is astonishing enough that he can declare that hypnotism should be discarded as a therapeutic weapon if it did not produce a completer cure than that recorded in Dr. Mason's case. By the same token I should give up using antacids because they do not cure peptic ulcer. But this is small matter for discussion when compared with his "criticism" of Drs. S. P. Hall-Smith and Alan Norton's excellent paper (August 23, 1952, p. 417). Dr. Lydon sneers at statistics, of which he admits little understanding, but his lack of understanding is much deeper. He suggests that it would be utter chaos in medicine if we accepted a patient's statement that "It's me nerves, doctor." Surely these very patients are the ones with stress situations who are ready to talk about them to a sympathetic physician who is not afraid to realize that all human beings are "psychosomatic" in structure, and hence that all medicine is psychosomatic. But perhaps Dr. Lydon does not believe at all in taking histories, but prefers to study his cases "objectively," to examine rashes with lens and microscope, never thinking of the human being (so aware) under the damaged skin. He declares the objective study to be the only scientific one. This is arrant and dangerous nonsense; neither doctor nor patient are objects, and the disease cannot be isolated from the human being who bears the signs and symptoms. Does Dr. Lydon think that the chemist's objective determination of a blood sugar value can tell one why it rules so unevenly high in an unhappy diabetic patient?

In this world of cold war and uncertain future, stress situations are all too frequent in individuals; disease is not necessarily produced, but such situations are often proved and major contributory factors to illness in many who cannot adjust themselves and where the strain is beyond the limits of their "elasticity." Will an objective outlook on our part help them, or should we perhaps ignore these patients, discarding them as mere neurotics, or would it be better perhaps if we trained ourselves, in all humility, to listen patiently while some at least purge themselves of their woes, some gain insight and strength to tackle their own indecisions, and some find in the doctor a brother to help and advise them? I for one, as a teacher of an older school, have not found it easy to learn a new discipline, but it has become essential, and I do know now that a stethoscope is often an obstruction to hearing.

It is not enough to study soma alone or psyche and soma together, for medicine is the study of the two as represented by man living in society. Words like psychosomatic or social medicine, therefore, describe incompletely the whole study. Of course smaller aspects must be isolated for investigation, even pure organic medicine if it can be found, but in such isolated studies it must not be forgotten that artificial separations from the whole matrix have been effected in order to limit the number of operative variables.

The spreading of tons of soothing greases on acres of neuro-dermatitis has had no more obvious success than many other therapeutic procedures practised by all of us, but surely these relatively poor results should make us only too ready to welcome a new understanding? I wonder if Dr. Lydon has ever given his patients phenobarbitone?

I hope he will not take my attack in a personal way—his views are not uniquely held—but I hope that before he replies he will read the gentle and human lectures by George Day and some of the writings of Walter Alvarez and Flanders Dunbar.—I am, etc.,

London, W.1. HUGH GAINSBOROUGH.

The Patient Tells His Story

SIR,—Dr. R. Logan (December 27, 1952, p. 1414) raises a number of points about which there is room for difference of opinion. He states, for example, that students and workers prefer a questionary type of "screening" to the "usual perfunctory medical inspection." In the first place it is questionable whether routine medical examinations conducted for whatever purpose need be perfunctory. Secondly, experience with questionary surveys conducted among undergraduates at the University of Wales does not suggest that students prefer this method to the personal interview. The usefulness of the routine examination is not, however, to be judged by the personal preferences either of student participant or medical officer. One of the most important functions of the routine examination—particularly when conducted among entrants to a university—is that it affords an opportunity for personal contacts to be made and for confidence to be gained in a service which the student may need subsequently during his academic career. Moreover, many entrants have minor anxieties which can often be allayed at the initial interview, and the importance of the examination as a medium for health education—the need for which is undoubted even at the university level—should not be overlooked. It is difficult to see how any of these functions can be carried out effectively by a questionary, however elaborate.

Questionary "screening" as a means of eliminating physical defects has obvious shortcomings, but it is perhaps particularly in the interpretation of emotionally charged symptoms that the questionary is found wanting. In any preventive and promotive scheme conducted among

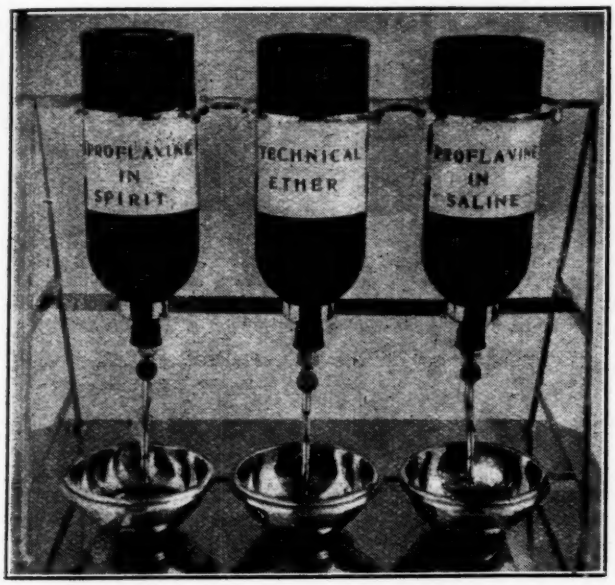
ostensibly healthy men and women examination should be sufficiently thorough to detect minor physical defects, and the interview should afford an opportunity for the discussion of psychological difficulties. It is often unspoken (and consequently unwritten) fears or hidden symptoms that are the most important, and personal medical interviews are still the best method for their detection. Questionaries should remain supplementary.—We are, etc.,

GRAHAM GRANT.
R. A. N. HITCHENS.

Cardiff.

Automatic Lotion Dispenser

SIR,—The lotion stand illustrated enables a constant and predetermined quantity of fluid to be delivered automatically into three stainless steel bowls without manual assistance. The sterile delivery units consist of cork, on- and off-tap, and delivery tube, the height of which above the bottom of the bowl can be varied by ¼ in. (2 cm.) according



to requirements. When the surgeon or assistant dips a swab into any bowl, the amount removed is automatically replaced from the inverted Winchester. The device has proved reliable, labour-saving, and economical. Thus the average saving of technical ether per week, in a theatre used for approximately 50 major operations per week, amounts to 17s. when compared with manual filling. Corresponding, but smaller, savings are also made in the use of proflavine.

Credit for the idea is due to Mr. R. A. H. Hill, senior theatre sister, who made the prototype model from plywood. The finished product was manufactured for us by Messrs. A. L. Hawkins, Ltd., 15, New Cavendish Street, London, W.1. I am indebted to Mr. T. Back of that firm for his suggestions in putting the idea into practice, and to Mr. E. A. Sheppard, of Oldchurch Hospital, for the photograph.—I am, etc.,

E. JAMES.

Rush Green Hospital, Romford Essex.

Dermatomyositis and Malignant Disease

SIR,—In his article on the skin manifestations of malignant disease (October 25, 1952, p. 911), Dr. L. Forman quotes some cases in his own experience of dermatomyositis occurring with malignant disease and refers to others in the literature. In an earlier report on a woman with a past history of carcinoma of the ovaries who developed dermatomyositis some years later, Dr. Forman[1] expressed the opinion that the relationship between the two diseases is not fortuitous.

This view is supported by the recent findings of Curtis, Blaylock, and Harrell,[2] who have reviewed the records of 45 cases of dermatomyositis since 1934, and report that eight of these patients, seven of whom were females, had some form of malignant disease. Carcinoma of the breast was present in two cases, but otherwise there was no uniformity in the type of malignancy. The onset of the dermatomyositis preceded the appearance of the malignant disease in all eight cases by an average of 18.6 months. Clinical improvement was observed in the dermatomyositis in six of the cases following treatment of the neoplasm. On the other hand, there was apparently no exacerbation of the dermatomyositis as the neoplasm progressed or metastasized.

I would like to record briefly a case of dermatomyositis together with malignant disease, in which the symptoms of dermatomyositis appeared approximately 14 months after operation for ovarian carcinoma.

The patient was a woman aged 63, with a florid rosaceous complexion and hypertension (B.P. 220/110) but no evidence of cardiac failure. In April, 1950, total hysterectomy and bilateral salpingo-oophorectomy were performed for carcinoma involving both ovaries. Complete removal of the growth was not possible because of secondary deposits in the posterior vaginal wall and rectum. Sections showed an ovarian carcinoma of relatively low local invasiveness, much of the tumour being in the form of pleomorphic adenocarcinoma, while in parts it was very suggestive of granulosa cell origin.

The symptoms of dermatomyositis appeared suddenly while she was on holiday at the seaside in June, 1951, and resembled acute sunburn, the exposed parts of the face, neck, arms, and hands becoming swollen and inflamed, and her eyes being almost closed with oedema. A few days later she found difficulty in swallowing, first solid foods, and then also liquids, and there was nasal regurgitation. In addition she became progressively weak, being at first unable to raise her arm to do her hair, and later to sit up from a recumbent position or to get in and out of bed without assistance.

She was readmitted in August, 1951, with low-grade pyrexia, profound myasthenia, total inability to swallow, and an erythematous dermatitis involving the face, neck, upper part of trunk, hands and arms, and, to a lesser extent, the legs up to the knees, with pitting oedema in some areas. A hard fixed mass was palpable in the pelvis. During the next few months the dermatomyositis regressed considerably, the dermatitis subsiding into a telangiectatic erythema with atrophy and pigmentary changes, while recovery of power occurred in some muscles. The weakness of the pharyngeal muscles, however, persisted, and she had to be fed continuously through an intra-nasal gastric tube. Death occurred due to bronchopneumonia in February, 1952. Post-mortem examination revealed a terminal generalized tuberculosis in addition to carcinomatosis and degenerative changes in the skin and muscles characteristic of dermatomyositis.—I am, etc.,

E. W. PROSSER THOMAS.

London, W.1.

[1] *Proc. roy. Soc. Med.*, 1951, **44**, 880.
[2] *J. Amer. med. Ass.*, 1952, **150**, 844.

Intussusception Associated with Trauma

SIR,—Donhauser and Kelly[1] have analysed 665 cases of intussusception in adults published in the English and American literature between 1900 and 1947. Of these, only one was associated with trauma. The following is a report of a similar incident occurring in a child.

A male child, aged 3½, was knocked down by a motor truck and was admitted to hospital immediately. He had extensive bruising of the abdominal wall, right hip, and thigh in addition to lesser abrasions and contusions on his limbs. He complained of pain in the right side of the abdomen, which was tender. There was a moderate degree of shock, his pulse rate being 120 per minute. Injury to the liver was suspected and he was kept under observation. His pulse rate remained steady for two hours but then rapidly rose to over 160 per minute. No change in his abdominal signs was noted, bowel sounds being present, and no free fluid could be detected. Liver dullness was normal.

Despite liberal fluid and colloid replacement his pulse rate remained very high. Four hours later bowel sounds could not be heard and the abdomen was explored. There was no vomiting. At operation a subserosal haematoma was found to have ruptured the peritoneum on the lateral wall of the caecum. A thin film of blood was present in the paracolic gutter. Bleeding had ceased. No other injury was found, but he had an intussusception of the jejunum situated 4 ft. (1.22 m.) distal to the duodeno-jejunal flexure. It was 3 in. (7.6 cm.) in length, reduction presented no difficulty, and there was no local predisposing factor present. The abdomen was closed and recovery was without incident.—I am, etc.,

Kingston-on-Thames. A. A. MURRAY.

REFERENCE

[1] *Amer. J. Surg.*, 1950, 79, 673.

Thrombocytopenic Purpura in Successive Pregnancies

SIR,—I have read with much interest Dr. G. T. Hammond's report (December 20, 1952, p. 1340) of a case in which thrombocytopenic purpura occurred in two successive pregnancies. In my own case reported in the *Journal* (December 11, 1948, p. 1020) there was a complete restoration to normal of the blood picture after the pregnancy, but in a subsequent pregnancy a year later a purpuric rash appeared early; the pregnancy ended in an abortion following a fall at home at 14 weeks. In a second case the platelet count and haemoglobin were apparently so adequate that transfusion was not carried out. There was little bleeding from the uterus following a normal delivery, but an episiotomy had been carried out and this gave rise to severe bleeding which proved very difficult to control. In this case also the infant showed thrombocytopenia. In both mother and child the blood picture was normal a month after delivery. In a third case where purpura was observed in two successive pregnancies a central placenta praevia demanded caesarean section in the second pregnancy. During this operation the control of haemorrhage presented great difficulty.

From these cases two points of interest emerge. First, the recurrence of purpura in successive pregnancies indicates some special constitutional factor present during pregnancy which leads to thrombocytopenia and which in some instances passes the placental barrier to affect the infant also. Once the pregnancy is over restoration to normal occurs, since presumably the factor ceases to operate, but recurrence in the subsequent pregnancy has been observed so frequently that there seems to be little doubt that there is a special constitutional tendency to purpura during the pregnancies of certain women. Secondly, the arrest of uterine haemorrhage is largely achieved by muscular activity, and therefore uterine bleeding is not a particular danger in these cases. It is in lacerations, caesarean section incisions, or episiotomy that the danger lies, and these are therefore to be avoided if at all possible. I would endorse Dr. Hammond's view that blood transfusion as near as possible to the onset of labour should be practised in every case where purpura is found in association with a reduction of the thrombocyte count.—I am, etc.,

Worcester. J. A. CHALMERS.

Radio-iodine and Thyroid Carcinoma

SIR,—We have no adequate explanation for the findings in the case of thyroid carcinoma (November 22, p. 1115) to which Dr. Ansell refers (December 20, p. 1358). His valuable suggestion that radio-iodide may have been concentrated and discharged without organic synthesis appears to conflict with the following observation, which we did not publish. The mean time of radio-iodide circulation for the relevant dose in this patient, as judged by initial urinary excretion, was 1.3 days and thus rather shorter than the mean retention time of radio-iodine in the tumour, which was 2.3 days.

Our suggestion of organic synthesis without follicular storage would explain some delay although perhaps not so large a one; but it would not account for the low protein-bound radio-iodine concentration observed in the plasma unless the synthesis did not proceed normally to thyroglobulin or to thyroxine, and of this we have no evidence. Nor could the facts be explained by Seidlin's observation[1] that radio-iodine may be concentrated in necrotic material remaining after destruction of thyroid carcinoma, since the dose in question was a test dose following thyroidectomy but preceding any radio-iodine therapy.

We have observed other patients more recently with such rapid radio-iodine "turnover" in thyroid carcinomata. We wished to draw attention to this type of case as being important although unexplained, since the opportunities for their treatment might be overlooked by using the normal methods of investigation.—I am, etc.,

London, W.C.1. E. ERIC POCHIN.

REFERENCE

[1] *J. clin. Endocr.*, 1949, 9, 1122.

Otitis and Ear Syringing

SIR,—Surely Mr. A. Tumarkin (December 20, 1952, p. 1361) is not serious in his gratuitous and rather patronizing advice to his general-practitioner colleagues that the syringing of ears should now be considered to be outside their competence? So far as recruiting boards are concerned he may be assured that the members of these boards are quite alive to the importance of an adequate ear examination, but I feel that the ideal conditions which he so desires, when "nothing shall be missed," will only be attained when E.N.T. consultants take their place among the members of the board, at a remuneration of approximately a guinea an hour. Until that time we must do our poor best.

Mr. Tumarkin further suggests that our hospital E.N.T. department should do our syringing for us. I do not expect that the consultant would himself do our 40-odd cases per week. I imagine that the job would be delegated. Those of us who have been doing this job regularly for six years and more feel that we are quite as competent with the syringe as any member of the hospital staff, apart from the consultant himself.

Would Mr. Tumarkin care to tell us how many cases of otitis or perforation which he has seen were due to the removal of wax by practitioners on the medical boards of the area for which he is consultant?—I am, etc.,

Leicester J. K. M. DEAS.

Sarah Gamp's Pharmacopoeia

SIR,—The other day, after manœuvring some gritty tablets down an inflamed pharynx, I consoled myself with reading *Martin Chuzzlewit* (Dickens being to the fore these days—see the *Journal*, November 8, 1952, p. 1039). I found that Sarah Gamp and her colleague Betsey Prig were in the habit of giving their patients "slime draughts." The actual passages are as follows: "'The physic and them things is on the drawers and mankleshelf,' said Mrs. Prig cursorily (going off duty and handing over to Sarah Gamp). 'He took his last slime draught at seven. The easy-chair an't soft enough. You'll want his piller.'" And: "Mrs. Gamp shook her head mysteriously, and pursed up her lips. 'There's fevers of the mind,' she said, 'as well as body. You may take your slime drafts till you flies into the air with efferwescence; but you won't cure that.'"

A slime draught (spelt "draft" in the second passage) was the sort of treatment which evidently appealed to Mrs. Gamp and her school. It may, however, have possessed some virtues, and must have been easy to swallow. I wonder if any of your readers have any information about this long-discarded prescription of the pharmacopoeia. What was its action, and what were its ingredients, which produced the "efferwescence" so vividly described by the eloquent "Sairah"?—I am, etc.,

Edinburgh. JAMES A. ROSS.

POINTS FROM LETTERS

Danger of Antihistamine Drugs

Dr. I. ROXON-ROPSCHITZ (Brisbane) writes: While I have no new clinical information to offer, I would like to report a case similar to that of Dr. W. O. Pitt (September 27, p. 724). In his case the ingestion of antihistaminic tablets was followed by an attack of rigor, nausea, and malaise. Ultimately after a few attacks of this kind (always following the intake of antihistaminics) his patient met with death. I was fortunate enough to connect similar attacks in my case with the intake of the antihistaminic methapyrilene hydrochloride (" diatrin ") before serious damage was done. To exclude the fallacy of *post hoc ergo propter hoc* I asked the patient after a couple of weeks to repeat once more the dose of methapyrilene hydrochloride and again fever of 102° F. (38.9° C.), rigor, vomiting, and general malaise reappeared. Recovery followed after three days.

Birth Times

Dr. L. B. CANE (Bungay) writes: The statistical table published by Dr. A. S. Simpson (October 11, p. 831) supported the popular idea that more babies arrive by night than during the day. In his review 55.5% of deliveries were between 11 p.m. and 11 a.m., and 44.5% between 11 a.m. and 11 p.m. In 1911 I published a similar table giving the number of births in each of the 24 hours, with totals for six-hourly periods, based, however, upon a smaller total of cases attended in our own practice. A later analysis from our records of the 2,000 deliveries confirms that most births occurred during the last half of the night, and fewest in the afternoon. In this series of 2,000 the percentages were: *Day*, 8 a.m. to 2 p.m., 25.14%; 2 p.m. to 8 p.m., 21.38%; total, 46.52%. *Night*, 8 p.m. to 2 a.m., 25.48%; 2 a.m. to 8 a.m., 28%; total, 53.48%. This analysis, like that of Dr. Simpson, indicates the preference for entering life in the period described by Burns as " the wee short hours ayont the twal."

Rotten Teeth

Dr. W. W. YELLOWLEES (Aberfeldy, Perthshire) writes: It is clear from the report of the Subcommittee of the Scottish Standing Dental Advisory Committee (October 25, p. 928) that for the control of dental caries the preventive dental services of this country are less advanced than those of prehistoric man. This conclusion is justified by the results of surveys revealing the excellent dental health of certain isolated, self-supporting peoples, living in areas without access to foods produced by the industrial activities of modern civilization. Is it not strange that, at a time when not only dental caries but also degenerative diseases of all kinds are rife and increasing, the medical professions of western civilization should condone the disastrous dietary habits of our time, and should make no protest against the intensive processing and refining which robs our food of much of its value before it reaches our tables . . . ? If we are to survive as a vigorous nation, we cannot afford to continue developing our machines to the neglect of our land. Successive governments have placed emphasis on industrial production for our survival and to-day we are choosing television sets rather than fresh butter; if this choice continues rotten teeth will continue rotting. The vital relationships between nutrition and health have been elucidated by many workers since the beginning of this century. Our profession has paid little heed to these relationships, and has concentrated on the study of the end-results of disease and degeneration. . . . If we are to " stop the rot " we must surely return to full-scale cultivation of our own soil. There is no other way, for without fresh whole food all other measures, however costly, will be in vain.

Asthma in Childhood

Dr. F. A. LOMAX (Kroonstad, S. Africa) writes: Dr. Cyril Josephs's letter on asthma (October 18, p. 881) prompts me to give my views on asthma derived from personal experience of the disease. Asthma is primarily an allergic disease. It only occurs when the potential asthmatic is being exposed to the allergen to which he is susceptible. Emotional disturbances and suggestion can only precipitate an attack of asthma in a person who has already been exposed to the allergen to which he is susceptible. Favourable emotions and states of mind cannot protect a potential asthmatic if he is exposed to a high concentration of the allergen to which he is susceptible. The commonest allergens which produce asthma are feathers, house dust, and grass pollen. Asthma occurs chiefly at night because the potential asthmatic sleeps for many hours on a pillow full of feather dust which may be a veritable poison to his sensitive respiratory tract, and because he sleeps in a room which is often full of house dust. In hospital potential asthmatics are often free of bronchial spasm because the pillows are not very old and the wards are exceptionally free of dust. To discover why an old feather pillow is so harmful to a potential asthmatic sensitive to feathers, cut open an old feather pillow and you will be amazed at the excessive amount of feather dust in it. My sympathy goes to the unfor[illegible] asthmatic whose attacks are brought on by an allergen b[illegible] treated as a psychological case. The best cure for asthma is to remove the responsible allergen from the patient or the patient from the allergen. If this can be done, the potential asthmatic will be free of asthma in spite of worry, anxiety, fear, annoyance, frustration, or any other emotional or mental disturbance, which some claim to be the primary cause of asthma.

Cancer of the Lung and Plastic Dentures

Dr. W. ST. A. F. HUBBARD (Penzance) writes: Mainly because a sudden increase in the incidence of carcinoma of the lung has more or less coincided with the much increased consumption of cigarettes, the latter have had to take most of the blame. It is true that other causes have also been suggested, such as exhaust gases and tarred road surfaces, but nowhere have I seen it mentioned that this increased incidence has occurred almost *pari passu* with the introduction of the new plastic materials for dentures in place of vulcanite. I was on the medical boards for ten years, and I remember having come across a dental mechanic with a peculiar dermatitis of the palms of his hands. He told me that the condition was not uncommon in those who have to make modern dentures. Whether, if what he told me is correct, such a dermatitis is due to some irritant property in the material itself or due to the handling of hot moulds, I do not know, but since then I have always felt a little suspicious about my own denture. It seems very unlikely that the plastic material is carcinogenic in ordinary use, but is it not possible that it may become so upon contact with hot tobacco smoke ?

Self-demand Feeding and Overfeeding

Dr. MURIEL C. GOODCHILD (Kettering) writes: I was extremely interested in the articles by Dr. Ian G. Wickes and Drs. J. D. Hay, P. D. Moss, and M. M. P. Ryan (November 29), I have been advocating appetite feeding in my clinics for some years, and have never found any baby suffer thereby. As to overfeeding, it does not, I think, occur but very, very rarely, the only symptom being occasional vomiting, the baby bringing up the surplus. The infant in these cases gains well and gives every appearance of being a healthy baby with firm muscles and good colour. . . . Every baby has to be treated as an individual in its feeding, and generalization without reservation is dangerous. One infant will gain far beyond the average ounce a day in the first three months on a quite inadequate feed " according to the books," while another will fail to gain more than half this amount on what would appear far too much. Both infants may well be healthy and well-favoured children. The really fat babies are usually not overfed by all the usual standards, and seem little the worse for their fat, which mostly drops off quite quickly as soon as they start to walk.

The Patient's Verdict

Dr. W. R. E. HARRISON (Buxted, Sussex) writes: Lord Moran's Harveian Oration (*Journal*, October 25, p. 936), and the admirable dissenting letters from Drs. R. W. Cockshut (November 1, p. 993), D. Hardie (November 22, p. 1151), and A. Piney (December 6, p. 1258), put in a nutshell the cruel dilemma of our time—the necessity for control versus the indispensability of freedom. What a change in medicine since my student days, when, apart from the glorious craft of surgery, all we had in the way of treatment was the elegant placebo of the *British Pharmacopoeia*. How easy then to follow the tenets of Hippocrates. But now with modern drugs we can keep old folk stretched a little longer on the rack of time. Antibiotics can stamp out the winnowing epidemics from the vast breeding-grounds of the East, and reduce its infant mortality to vanishing point. " Social medicine " and " prevention " can secure for a time the survival of the poorest genes. Soon we may be able to prolong the span of life beyond the ranges of Methuselah, and determine the sex of children yet to come. But overshadowing and determining all is the spectre of over-population—that fearful geometrical progression which must be checked quite soon or bring the human species to a cataclysm of starvation. Can anything be done to cut this vicious spiral ? Can " controls " do it ? Apparently not—not even when implemented by the secret police and hellish concentration camps. Can freedom (i.e., self-control) do it ? Not, surely, among the " under-developed " masses ? Yet many may still feel that medicine with its record of wisdom and knowledge of men might at least propound the problem personally through its practitioners and in print and through the radio, so that our patients would know what to expect, understand the danger, and, being forewarned, might conceivably meet and overcome it.

Obituary

JAMES COLE MARSHALL, M.D., F.R.C.S.

We announce with regret the death of Mr. J. Cole Marshall, the distinguished ophthalmic surgeon, on December 24, 1952, at the age of 76.

James Cole Marshall was born in 1876 at Blandford, Dorset, the eldest son of James and Emily Marshall. He received his medical education at St. Bartholomew's Hospital Medical School, where he qualified with the Conjoint diploma in 1900. In the following year he took the London M.B. For a time he was interested in gynaecology, and his M.D. degree, which he took in 1904, a year after taking the F.R.C.S., was in this subject. Soon afterwards, however, he was attracted to ophthalmology and decided to specialize in it.

In the course of his career Cole Marshall held honorary appointments at several hospitals, including the Royal Waterloo, Lambeth L.C.C. (for many years), the Northwood and Pinner, and from 1913 to 1946 the Western Ophthalmic. There he was senior surgeon for eleven years and did his main work. Among other appointments he was honorary oculist to the Royal Academy of Music and to the Royal London Society for Teaching and Training the Blind. During the first world war he served in the R.A.M.C. as ophthalmic specialist to the Rhine Army.

Cole Marshall was an efficient all-round ophthalmic surgeon, deliberate, careful, and skilful as an operator, sound in clinical judgment, and thorough in the examination of his patients. He was one of the first in Britain to practise the modern operative treatment of retinal detachment, which he continued to study with increasing enthusiasm and practise with remarkable success for the rest of his professional life. This success was primarily due to his careful and prolonged survey of the fundus for retinal tears, which were then systematically plotted on charts that he had devised for the purpose and which are still in use. His energy and capacity for work in this and other respects were outstanding, and his methods an example and a training not only to a long succession of house-surgeons and registrars but also to his colleagues. It is no exaggeration to say that he founded a school of detachment surgery at the Western, where he operated upon an astonishing number of cases, and attracted patients from all over the British Isles and surgeons from abroad as frequent visitors. Although he tried out most of the various techniques devised from time to time, ultimately he remained faithful to puncture diathermy, which he considered preferable to surface coagulation. As senior surgeon and chairman of the medical committee he combined calm common sense with considerable administrative ability. His attitude to his hospital patients was admirable. Everyone was accepted on terms of equality and treated with great kindness and respect. Altogether, Cole Marshall was a well-balanced and lovable personality, affectionately admired by his patients, by the staff of the hospital, and by all his colleagues. In 1935 he was the Middlemore lecturer at the Birmingham Eye Hospital, and in 1938 Hunterian Professor at the Royal College of Surgeons. His book on *The Operative Technique of Retinal Detachment* was published in the following year.

Endowed with a delightful and charming personality and a keen sense of humour, Cole Marshall had many interests outside his profession—for example, in music and painting, in both of which his judgment was excellent. Between the wars he built a most attractive house and developed a beautiful garden in Rickmansworth. The garden was a perennial source of pleasure in that he could there enjoy the twin delights of gardening and bird-watching—two favourite hobbies.

In 1905 he married Miss Margaret Compton, who, with their three daughters, survives him. He was singularly fortunate in a happy home life. To the end he looked much younger than his years, and during the last year or two had recovered to a great extent from an illness which had precipitated a premature retirement from private practice. He never lost his interest in the affairs of the Western Ophthalmic Hospital, where his death has been felt by all as the personal loss of a friend.—A. R.-G.

GIBBON FitzGIBBON, M.D., M.Ch., F.R.C.P.I., F.R.C.O.G.

Dr. Gibbon FitzGibbon, who died on December 1, 1952, at Clonskeagh, Dublin, at the age of 75, was Master of the Rotunda from 1919 to 1926.

Gibbon FitzGibbon was born in Dublin in 1877, the son of Henry FitzGibbon, visiting surgeon to the Royal City of Dublin Hospital and a past President of the Royal College of Surgeons in Ireland. The FitzGibbon family had long been prominent in Dublin, especially in the legal profession, one of the most famous of Irish lawyers being Lord Clare, of whom it was written, "No individual Irishman has ever exercised a greater authority over the affairs and fortunes of his native country." Gibbon FitzGibbon was educated at Trinity College, Dublin, and graduated M.B., B.Ch. in 1900. The Boer War was then in progress, and he volunteered and served with the R.A.M.C., gaining experience in war surgery and typhoid fever. On returning he wrote a thesis on typhoid fever, proceeding M.D. in 1901. In this connexion he advocated the use of early mobilization in convalescence, a therapeutic measure which he introduced in the puerperium and post-operative routine. He was elected F.R.C.P.I. in 1910 and F.R.C.O.G. in 1929, being a Foundation Fellow of the latter College. In 1930 he obtained the M.Ch.

FitzGibbon became Assistant Master at the Rotunda in 1902, to Purefoy, and completed his term of office in 1905—a period in which the standard of abdominal surgery was rapidly improving. In 1910 he was appointed gynaecologist to the Royal City of Dublin Hospital. During the subsequent years he took an active part in the obstetric section of the Royal Academy of Medicine in Ireland, becoming president in 1915.

In 1919 FitzGibbon succeeded Jellet to the Mastership of the Rotunda. During his period of office he proved himself to be an original and courageous teacher. His investigations in the treatment of disproportion are well remembered, and those who were fortunate enough to be his assistants will all testify to the thorough trial of labour that was given to his patients. A trial of labour in FitzGibbon's hands was preceded by a careful assessment and a definite prognosis, which were rarely at fault. It was an education to see his careful delivery of a patient with the obstetric forceps. He advocated induction of labour to avoid the dangers of disproportion; the method he used at first was the introduction of a stomach tube, but later he recommended and practised puncture of the membranes. In 1923 his textbook,

Practical Midwifery, was published. It is a very practical and personal account of his views on obstetric problems.

FitzGibbon's Mastership coincided with the period when the financial problems associated with hospital administration were becoming exceedingly difficult. He made every effort to support economy, and was always an outspoken critic of the comparative extravagance that has since characterized hospital expenditure.

Ireland in the years following the 1914–18 war was disturbed by civil strife, and street conditions in Dublin were often unsafe because of clashes between the Black-and-Tans and the I.R.A. At times there were no students to brave the disturbed city at night, and it is recorded that FitzGibbon was the only Master to have attended a normal case in the patient's own home, the rule of the hospital being that if there is no other to go it is the Master's responsibility.

When FitzGibbon completed his Mastership he became gynaecologist to Mercer's Hospital until he retired from practice in 1951. When the British Medical Association met at Edinburgh in 1927 he was a vice-president of the Section of Obstetrics and Gynaecology, and in 1929 he was President of the Seventh International Congress of Obstetricians and Gynaecologists.

Gibbon FitzGibbon was held in respect and affection by his colleagues and pupils, and, if his criticisms were outspoken, they were sincere; when opinions differed frank discussion usually resulted in the development of mutual understanding. His interests were wide and included shooting and fishing. He was full of resource, as was instanced some years ago when he saved a friend's life by improvising a tourniquet when a shooting accident had resulted in the severing of the axillary artery. He was a member of the Church of Ireland and attended St. Patrick's Cathedral regularly. The large attendance at the funeral service in the cathedral was a testimony to the affection and respect in which he was held by the people of Dublin.

In 1900 he married Lilian, the daughter of William Stoker, professor of surgery in the Ledwich School. Mrs. FitzGibbon, who died in 1947, was held in high regard by all and was a charming hostess to those who visited the Master's house. There are three children of the marriage, two sons and a daughter. The sons, Gibbon and Henry, both served in His Majesty's Forces in the recent war. All three are married and have children.

We are indebted to Mr. G. W. THEOBALD for the following appreciation: Dr. Gibbon FitzGibbon, known to all and sundry as Fitz, hid a shy, reserved, and sensitive nature under a brusque exterior—which was sometimes construed as rude. He had wide interests, possessed a strong mechanical bent, and was clever with his fingers. When his children were young he devised intricate electrical railway systems, and during the lonely years following the death of his wife he found solace in repairing watches.

FitzGibbon was one of the outstanding obstetricians of this century. He introduced both the term and the concept of "trial labour," and read three lectures on this subject at the invitation of the University of London. He was probably the first to attempt the expectant treatment of placenta praevia (as early as 1920), but was forced to abandon the method because of the absence of any blood transfusion service. He was, moreover, one of the early champions of induction of premature labour by puncturing the membranes. FitzGibbon was a pioneer in the treatment of uterine prolapse by vaginal hysterectomy, and devised possibly the best operation for the treatment of this condition in young women. This operation consists essentially in shortening the parametric tissue without amputating the cervix. While serving as a volunteer during the Boer War he became interested in the early ambulatory treatment of typhoid fever, and was always in favour of getting obstetric and gynaecological patients out of bed as soon as possible after the baby was born or the operation was over.

FitzGibbon contributed much and wrote little, but his ideas live through the teaching of his disciples. He was a punctilious Christian gentleman, and lies buried close to the cathedral in which he loved to worship. Those who were privileged to know him loved him, and they will miss the merry twinkle in his very bright blue eyes, sometimes unashamedly dimmed with the tears of emotion, and they know that they will not meet his like again.

Major-General H. ROTHERY NUTT, M.D., F.R.C.S. I.M.S.(ret.)

We record with regret the death on January 2 of Major-General H. Rothery Nutt, I.M.S.(ret.), at his home in Eastbourne a few days after he had completed his 76th year.

Harold Rothery Nutt, the son of William Henry Rothery Nutt, was born on December 28, 1876, and received his general education at Bedford Modern School. For his medical education he entered the medical school of St. Mary's Hospital, London, and qualified with the Conjoint diploma in 1900. In the same year he graduated M.B.Lond. and in 1908 proceeded M.D. After qualification Nutt became a house-surgeon at St. Mary's, and then entered the I.M.S. in June, 1901. He had always been an outstanding student, passing his primary fellowship examination in the course of his student career and for a time holding the appointment of assistant demonstrator in physiology. He was able to take his final F.R.C.S. in 1902 when he was only 25.

In India Nutt performed the necessary early years of duty with the Indian Army that fell to the lot of every young I.M.S. officer, and in 1908 he entered the civil side of the Service, being appointed to the United Provinces. Here he acted as civil surgeon in various areas of those provinces, and in 1909 married the lady who has been his lifelong partner. When war came in 1914 Major Nutt, as he then was, was recalled to military employ and spent some time with hospitals in Egypt and also in hospital ships. At the end of the war he reverted to civil employ and in 1920 was appointed professor of surgery at the Lucknow Medical College and surgeon to King George's Hospital, Lucknow. In 1921, on the formation of Lucknow University, he became the professor in surgery there.

As a surgeon Nutt was characterized by his soundness and careful attention to detail. But he preferred the more diverse work of a civil surgeon to the academic life, and after four years with the medical college he obtained a large civil surgeoncy, and later completed his clinical career as civil surgeon of Allahabad. In 1930 Lieutenant-Colonel Nutt was promoted full colonel and appointed Inspector-General of Civil Hospitals, United Provinces, where he continued for three years showing the same thoroughness in his administrative duties as he had in his surgery. During 1931–2 he served as President of the United Provinces Branch of the B.M.A.

In 1933 Colonel Nutt was promoted major-general; he further had the honour of being appointed an Honorary Surgeon to the King and became Surgeon-General to the Government of Bombay. However, in Bombay he suffered at times from poor health and retired from the Service in 1935.

On retirement Major-General Nutt, having built a house in Bexhill, devoted himself to gardening, which, together with motoring, was his chief hobby. After the second world war he moved to Eastbourne and continued to work on boards under the Ministry of Pensions. Harold Nutt was always a cheerful and witty companion, and his wife was his devoted comrade for over 43 years. To his wife and to their married daughter we offer our deep sympathy.—C. A. S.

SIR T. DRUMMOND SHIELS, *M.C.*, M.B., Ch.B.

We announce with regret the death in London on January 1 at the age of 71 of Sir T. Drummond Shiels, formerly Member of Parliament for Edinburgh East.

Thomas Drummond Shiels was born at Edinburgh on August 7, 1881, the son of Mr. James Drummond Shiels, a lithographer, and, after a board-school education, he was trained as a professional photographer, subsequently joining his father in a photographic business in Edinburgh. In the war of 1914–18 Shiels held a commission in the Royal Scots and later commanded, with the rank of captain, a trench mortar battery of the 9th Scottish Division. He was severely wounded at Arras in 1917, was mentioned in dispatches, and was awarded the Military Cross and the Belgian Croix de Guerre. After the war, and because he was keenly interested in public health, he enrolled as a medical student at the University of Edinburgh and graduated M.B., Ch.B. in 1924.

Shiels was a past president of the Edinburgh Fabian Society, and he became a member of the Edinburgh town council in 1919. He stood for Parliament in the year in which he graduated, 1924, and was returned as the Labour Member for Edinburgh East. He retained the seat at the 1929 General Election but was defeated in 1931. As a Member, he carried out his Parliamentary duties with zeal and conscientiousness. He was a member of the 1927 Royal Commission on Constitutional Reform in Ceylon; in 1929 he was appointed Parliamentary Under-Secretary for India; and in 1930–1 he was employed in a similar capacity at the Colonial Office.

He did much good work as chairman of the Joint Standing Committee for Educational Work of the Non-political Empire Societies in London. He had a wide knowledge of African problems and showed much sympathy towards Africans, and, indeed, towards all coloured people, in their desire for intellectual advancement. As a member of the council for many years and as a vice-president of the Royal Empire Society and chairman of its library committee, its information bureau, and its colonial group, his efforts produced good results. He was also chairman of the Dominions, India, and Colonies Panel of the British Film Institute, and in 1939–40 he was a member of the Dominions Committee of the British Medical Association. His work was recognized in 1939, when he was created a knight.

After losing his seat in Parliament Shiels became deputy secretary of the Empire Parliamentary Association, under Sir Howard d'Egville. During the last war he was acting secretary of the United Kingdom branch of the association. He became a member of the Colonial Development Council in 1946 and in the same year he was appointed public relations officer to the Post Office, holding the latter post until 1949, when he retired. Also in 1946 he became a member of the committee appointed by the Prime Minister to consider, from all aspects, the future of the terraces adjoining Regent's Park in London. As medical secretary of the British Social Hygiene Council (now the British Social Biology Council) from 1933 to 1941, and later as medical adviser and vice-president, Shiels did much to further the work of the council. Always interested in Colonial and Empire affairs, he was the author of a number of publications, and recently he was responsible as advisory editor for the compilation of *The British Commonwealth: Family of Peoples*, which was favourably reviewed.

Shiels was a man of ready sympathies. No call for assistance went unheard by him, and there are many in all walks of life who are grateful to him for help received or for his kindly intervention on their behalf. He had a keen sense of humour and a wit which never carried a barbed shaft. If he could, he would speak well of a person; if he could not, he would remain silent. He was twice married, first, in 1904, to Miss Christian Blair Young, by whom he had one daughter. His first wife died in 1948 and in 1950 he married Miss Gladys Buhler, who survives him, together with the daughter of his first marriage.

Dr. ARTHUR CANT, who was within a few days of his 80th birthday when he died on November 30, 1952, was a well-known general practitioner in the Midlands. Born on December 12, 1872, and educated at King Edward's School, Birmingham, he studied medicine at Mason's College, Birmingham, qualifying M.R.C.S., L.R.C.P. in 1895. He received the degrees of M.B., Ch.B., Birmingham, in 1901, when the university was granted its charter. After holding house appointments in Birmingham and Kidderminster, he settled in practice at Coleshill, Warwickshire, more than 50 years ago, and soon became not only the doctor but the trusted friend of a widespread rural community. Working to the end of his long life, Dr. Cant retained the affection of his patients to the last.

Dr. F. S. CLIFF died suddenly at Blackpool on December 3, at the comparatively early age of 46 years. His death came as a shock to his colleagues and friends in the Manchester region, and particularly in the Lancaster and Kendal group of hospitals, for he had a long and honourable record of service in that area. Frank Storey Cliff was a native of Lancaster, and was proud of his connexion with the Red Rose. He received his early education at the Lancaster Royal Grammar School, where he is still remembered by contemporaries as a keen scholar, as a sturdy rugby forward, and as an imperturbable cricketer. In his senior year he was head boy, and so good was his record of loyalty to his school that a few years ago he was elected to the board of governors. In 1924 he entered upon the study of medicine at University College Hospital, London, where he carried on the tradition of scholarship and sportsmanship which had characterized his years at school, playing both rugby and cricket for the hospital. He qualified in 1929, graduated M.B., B.S. in 1930, proceeded M.D. in 1932, and took the M.R.C.P. in the following year. His house appointments included those of house-surgeon at the Royal Surrey County Hospital and house-physician under Dr. Lewis at University College Hospital, where his association with Sir Archibald Gray gave him his first and lasting ambition to become a dermatologist. His last house appointment was in his native Lancaster, where he was senior house-surgeon at the Royal Infirmary for a time. With experiences and qualifications such as those one would have expected early specialization, but Frank Cliff wanted to learn medicine the sure way. He entered general practice as the physician in a large firm in Morecambe, where for many years he did much of the specialized medicine, and in a short time he was universally recognized as a man who could give a very sound opinion on all medical and dermatological cases. He was for many years physician on

the staff of the Queen Victoria Hospital, Morecambe, and the Royal Lancaster Infirmary, where latterly he had also a clinic in dermatology. During those years, his years of achievement, he served a community which will always be grateful for his work and which will always revere his memory as a fine physician. He brought to the provinces a standard of knowledge and skill equal to that of the great teaching school, and that he did deliberately, being inspired with the ambition to carry the torch of learning into places remote from the centres of education. In 1939 he joined the R.A.M.C. Supplementary Reserve, and went with the British Expeditionary Force to France as a major. He took part in the long and trying retreat from Belgium, and was among the many who were evacuated from the beaches of Dunkirk. With the advent of the National Health Service, Frank Cliff achieved his ambition to become a consultant dermatologist, and, while that was what he himself desired most of all, many of his colleagues thought that the gain of dermatology had been the loss of medicine. He served a large and scattered area stretching from Barrow to Blackpool, and including Kendal, Lancaster, and Morecambe, and throughout this wide area he made many friends among admiring colleagues and grateful patients. He will be sorely missed in Lonsdale, both north and south of the sands, and with his wide experience and encyclopaedic knowledge of two great specialties he will be a hard man to follow and one whom it will be almost impossible to replace.—J. McF.

Dr. THOMAS MACKENZIE, who died on December 5 at the age of 90, was born on April 18, 1862, and was educated at Larchfield Academy, Helensburgh, and at the University of Glasgow, where he graduated M.A. in 1882. He then transferred to the University of Edinburgh to study medicine, taking the degrees of M.B., C.M. in 1886, and proceeding M.D., with special commendation, five years later. He held the post of house-surgeon at Edinburgh Royal Infirmary, being a contemporary of the late George Lovell Gulland, who became professor of clinical medicine in the University of Edinburgh. After several years as an assistant in general practice, Dr. Mackenzie settled in Douglas, Isle of Man, where, among other appointments, he held those of surgeon to the Governor and commanding officer of the Isle of Man company of the King's Royal Liverpool Regiment. For his services to the Army he was awarded the Volunteer Decoration. He served in the war of 1914–18, and for several years was in command of the Cottonera Military Hospital in Malta. He retired from the Army with the rank of colonel, and then, having sold his practice in the Isle of Man, lived for a time in the Channel Islands. Voyages to Canada, Australia, and New Zealand followed, until he and his wife, formerly Miss Erie Turner, of Hamilton, Ontario, finally settled in Switzerland. Mrs. Mackenzie died three years ago.

The death of GEORGE TRUSTRAM WATSON on December 6 breaks a chain of five generations in the medical profession at Tunbridge Wells. He was the son of the late Dr. George Samuel Watson and younger brother of Dr. C. R. Watson; his mother was the daughter of Dr. Trustram, also a well-known practitioner in the same town. Born on October 9, 1871, at Thaxted, in Essex, he was educated at Tonbridge School, whence he won a scholarship to Peterhouse, Cambridge. After gaining first-class honours in the Natural Sciences Tripos he next obtained a scholarship at St. George's Hospital Medical School. Graduating M.B., B.Chir. in 1897, in which year he took also the M.R.C.S., L.R.C.P., he held the usual resident appointments at his own hospital, and in 1899 he obtained the F.R.C.S. All that, obviously, could have been the prelude to a surgical career and to Harley Street, but, after one year at Hastings, he joined his father and his elder brother in practice at Tunbridge Wells in 1901, and in the same year he married Miss Dorothy Rankin, who, with their two daughters, survives him. He quickly acceded to the surgical staff of the Kent and Sussex Hospital, where he was a tower of strength for many years. Both as surgeon and as general practitioner Watson enjoyed the highest reputation, not merely among his wide circle of patients as a wise counsellor and a real friend, but also among his colleagues as one who upheld the best standards of medicine and of professional ethics. No one ever knew him to do a mean or an unworthy deed and no one thought him capable of such a thing. He was a devout churchman who acted up to his principles and was rightly admired and respected by the whole town. During the 1914–18 war he served in the R.A.M.C. as a surgeon at the Herbert Hospital, Woolwich, and as assistant county director of the British Red Cross Society for Kent. For his services he was appointed O.B.E. in 1920. In 1936 he gave up operative surgery, but he found it more difficult to retire from general practice because so many of his old patients insisted on seeing him. His end was very sudden, though he had been warned some months ago that his hold on life was precarious.

Dr. EDWARD JAMES FOULDS, who died at his home at Greenmount, near Bury, Lancashire, on December 6, 1952, at the age of 56, was born at Colne on February 26, 1896, the youngest son of a well-known Quaker family. He was educated at Ackworth and Bootham, Yorkshire, before proceeding to the University of Manchester, where he graduated B.Sc. in chemistry, with honours. For eight years he resided at Dalton Hall, where he took part in many activities, football being his favourite game. After the war of 1914–18, in which he served with the Friends Ambulance Unit, he returned to the University and took the degrees of M.B., Ch.B in 1923. He then held the posts of house-surgeon at Manchester Royal Infirmary and at St. Mary's Hospital, Manchester, and assistant medical officer at Withington Hospital until 1925, when he joined and later succeeded Dr. A. P. Nuttall in general practice at Bury. He was appointed honorary assistant physician at Bury Infirmary in 1929, becoming a full member of the staff in 1936. He was responsible in large measure for the development and enlargement of the medical side of the hospital, one ward of which is to be named after him. In 1932–3 he was chairman of the Bury Division of the British Medical Association. For many years Dr. Foulds laboured under an increasing burden of ill-health, but he remained at his work, often under great physical discomfort, until his sudden final illness. He was a man of high moral standing and lived up to the strict code of his faith, but that did not suppress his humour. As a raconteur he was the joy of his friends and a stimulant to his patients. His caustic wit could quickly deflate pretentiousness, which was anathema to him. His patients, especially the old and lonely, experienced many kindnesses, done quietly and discreetly, to relieve them of anxiety and want. His knowledge of medicine was wide, and as a clinician he was very astute. In 1927 he married Helen Shirley Smith, and she survives him, together with a daughter and a son.—R. W. P.

Dr. HAROLD EDWARD RUTHERFOORD, who died at his home at Chichester on December 12, 1952, at the age of 80, was born in Calcutta on December 7, 1872. After a childhood spent in Ireland and an early education in England he entered Trinity College, Dublin, where he graduated M.B., B.Ch. in 1897, proceeding M.D. later in the same year. His stay at Trinity College, Dublin, was remarkable for his prowess at hockey, which in its modern form had been only recently introduced into Ireland. With his three brothers, he was a founder member of the Three Rock Rovers Hockey Club. In 1895 he played for Ireland against Wales in the first international hockey match between those two countries. During the following two years he played in six international matches. After graduating, Dr. Rutherfoord held resident posts, first at the Taunton and Somerset Hospital and later at the Bridgwater Hospital. In 1900 he became a ship surgeon, and spent two years travelling between England and the East. In 1903 he settled in general practice at Hatch Beauchamp, in Somerset. Five years later

he joined the late Dr. J. J. Blagden at Chester, where he stayed until 1915, when he moved to Chichester. In 1919 he was elected honorary physician to the Royal West Sussex Hospital, a post which he held until 1937. Two years later he retired from practice. Dr. Rutherfoord's retirement was an active one, and until the age of 76, when ill-health overtook him, he played lawn tennis regularly. He leaves a widow, a daughter, and two sons, the younger of whom is a general practitioner.

Dr. E. H. HUNT, who spent most of his professional life in India, died in London on December 15. Edmund Henderson Hunt was born in 1874 and educated at Harrow and Balliol College, Oxford. He took first-class honours in physiology, and worked for a year with the late Professor J. S. Haldane before going to St. Bartholomew's Hospital. He graduated M.B., B.Chir. in 1900, and after holding an appointment as house-surgeon at St. Bartholomew's he decided to take higher surgical degrees, gaining the M.Chir. in 1903 and becoming F.R.C.S. in the same year. In an appreciation recently published in *The Times* Sir Stuart Fraser mentions that it was while Dr. Hunt was working at the Radcliffe Infirmary at this stage of his career that he became seriously ill as a result of blood poisoning contracted from a patient. Out of four who got this infection he was the only survivor. In 1903 he went to India as chief medical officer of the Nizam State Railway in Hyderabad, and he retained this post for 28 years. He was president of the Hyderabad branch of the B.M.A. in 1925–6. While in India he developed a keen interest in archaeology, and some of his finds are now in the Pitt-Rivers Museum at Oxford. When he returned to this country in 1932 Dr. Hunt settled at Farnham, Surrey, where he developed many fresh interests, including bee-keeping. For a time he served on the committee of the National Honey Show. In 1904 Dr. Hunt married Laura Mary, the daughter of Sir James and Lady Buckingham. He leaves a widow, three daughters, and three sons, two of whom are well-known medical men, Dr. John H. Hunt and Mr. Alan H. Hunt.

Universities and Colleges

UNIVERSITY OF OXFORD

In Congregation on December 13 the following medical degrees were conferred:

B.M.—J. L. T. Birley, G. B. King, I. C. S. Normand, B. D. M. Williams, A. C. Allison, M. E. H. Barrow, J. O. W. Beilby, E. M. P. Brett, H. E. Green, D. B. L. Skeggs, J. Turpin, N. Ward, B. W. M. Macartney, B. Pigott, D. C. Robinson, J. R. B. Saxby, J. V. O. Reid, C. J. Dickinson, J. N. Doig, J. McC. Forrester, R. S. M. Ling, D. I'A. Alexander, R. D. Jones, D. J. Waters, P. J. Barber, J. D. W. Hunter, G. D. Horler, J. C. F. Johnstone, K. H. Lewis, E. J. W. A. O. Bowie, N. Howard, H. G. T. Bridge, M. R. P. Hall, R. H. G. Perks, D. D. Gibbs, W. Hift, R. H. Moore, D. A. S. Martin, J. Wharton, Mrs. Elisabeth M. Bell, Janet M. Marks, Josephine H. Smith, Hilary K. R. Steen, Cynthia M. Ward, Mrs. Margaret S. Barrett, Barbara J. Edwards, Gertrude M. Trevaldwyn.

UNIVERSITY OF CAMBRIDGE

The Vice-Chancellor of the University, Sir Lionel Whitby, Master of Downing College and Regius Professor of Physic, has been appointed a governor of Bromsgrove School until December 3, 1957.

Professor A. Leslie Banks, professor of human ecology in the University, has been appointed a governor of the United Cambridge Hospitals from March 31, 1953, for three years.

Professor V. B. Wigglesworth, F.R.S., has been appointed a Manager of the Michael Perkins Fund from January 26, 1953, for five years.

The Raymond Horton-Smith Prize for 1951–2 has been awarded to Dr. David Robertson Smith and Dr. James Paget Henry (equally).

The following Associate Lecturers have been appointed in the subjects indicated in parentheses: Mr. C. Parish (Thoracic Surgery), Mr. L. M. Rouillard (Plastic Surgery), and Dr. B. Donnelly (Radiology), all from October 1, 1952, for three years; and Dr. W. Paton Philip (Diseases of the Chest), from October 1, 1952, for one year.

The following degrees have been conferred:

M.D.—D. G. Vulliamy, P. H. Buxton, L. Wolman, J. Perrin, P. D. B. Davies.

M.CHIR.—R. N. Ticehurst.

M.A.—*Lady May Mellanby.

M.B., B.CHIR.—*Nancy I. Cardno, J. H. Newton, Mrs. Petrie Maud Newton, *H. Nichol.

M.B.—*Mrs. Svarna Lata Bhatia.

*By proxy.

The following candidates have been approved at the examinations indicated:

FINAL M.B. (Old Regulations).—*Part I (Surgery, Midwifery, and Gynaecology):* A. B. Aistrop, R. H. Champion, D. W. Couch, K. Coventry, R. J. Daniels, E. R. Davies, Mrs. E. A. Eldon, J. P. F. C. Evans, J. M. Fitzsimons, J. T. Fitzsimons, G. McC. Gransden, P. M. Hacking, P. W. Hayward, T. F. Hebblethwaite, M. B. Heywood-Waddington, S. M. Houghton, H. R. M. Johnson, B. P. Jones, R. V. Jones, A. J. Kenmuir-Evans, E. O'G. Kirwan, S. R. Laing, D. G. Lethem, H. E. D. Lloyd, I. B. Lyon, A. C. Markland, B. A. Maurice, R. H. Morgan, A. B. Myles, M. J. Mynott, H. G. Penman, M. J. Pritchard, P. H. Pullen, I. R. D. Reid, R. J. Sellick, G. D. H. Shephard, M. B. Teal, M. Trede, R. J. Vaughan, R. E. Williams, W. A. Williamson. *Part II (Principles and Practice of Physic, Pathology, and Pharmacology):* J. R. McNeill. New Regulations.—*Part II ([3]Principles and Practice of Physic, [2]Surgery, [4]Midwifery and Gynaecology)* (the figures indicate the section or sections passed): [3]C. W. M. Adams, [3]A. J. F. Barratt, [2 3 4]W. M. Beatley, [2 3 4]M. H. Bennett, [4]D. W. K. Bird, [2 4]J. S. Bland, [3]G. M. P. Boyes, [3]D. A. P. Burton, [2 4]V. G. Caiger, [3]D. Campbell, [2 3 4]W. H. Chapman, [4]H. S. Chen, [4]J. Cocker, [2 4]C. J. Cooper, [2 3 4]S. H. Dallas, [3]P. F. Dixon, [3]A. B. Donnison, [2]J. Fairley, [2 4]S. J. Fisher, [2 3 4]D. B. Fox, [3]Mrs. H. French, [3]A. E. Gibbs, [2 4]A. M. B. Golding, [2]R. K. Greenwood, [2 3 4]J. M. S. Grieve, [4]A. N. Griffith, [2 4]H. F. Grundy, [3]M. C. Guard, [2 3]A. M. Hammerton-Fraser, [4]R. D. Hernaman-Johnson, [2 4]M. D. Heyman, [2 4]A. F. Hignell, [3]C. B. Hobbs, [2 3]J. A. Holland, [2 4]V. M. C. B. P. Hollyhock, [2 4]B. L. Hoskyns, [4]R. J. Hutchinson, [3]B. S. Jay, [3]J. R. Kirkup, [4]H. E. Lichtenstein, [2]I. S. Lister, [2 3]J. S. Lloyd, [2 3]M. C. G. Mantell, [3]G. G. Matthews, [4]P. L. Maybury, [2 3 4]R. L. Morgan, [3]K. F. Morrice, [4]T. M. O. Morris, [4]M. T. O'Donnell, [4]A. B. L. Peake, [2 4]M. J. W. Penn, [4]A. O. Peters, [2 3 4]J. T. H. Pick, [4]J. F. Preece, [3]P. M. H. Rack, [4]G. L. S. Rankin, [4]F. Reiseger, [4]T. L. Ringrose, [2 3 4]R. D. W. Romanis, [2]W. A. G. Roper, [2 4]R. Routledge, [3]C. S. Savage, [2]T. L. Scully, [3]E. P. Simkin, [3]I. F. Smith, [2 3 4]E. J. Stacey, [3]I. A. Stewart, [2 4]G. D. Stones, [2 3 4]L. J. Stretton, [2 4]M. C. Thornton, [4]D. S. Trouton, [3]J. A. M. Tudor-Hart, [3]A. E. Wagstaff, [3]H. C. M. Walton, [3]A. W. Warltier, [4]P. A. Watt, [3]I. Weinbren, [3]Mrs. P. M. Whitehouse, [3]W. G. Williams, [3]S. M. Worrall, [2 4]M. T. C. Wright. *Part I (Pathology and Pharmacology):* I. M. Allison, J. D. Avison, L. P. Balfour-Lynn, S. G. Bayliss, M. H. Bennett, Mrs. A. B. Bentinck Speed, R. J. Blow, D. B. Bower, T. C. Bradford, J. A. D. Briggs, I. M. Brown, L. F. Brunt, H. M. Buckland, L. R. L. Burton, J. B. Carver, Mrs. A. Clarke, J. M. Clifford, G. M. Cochrane, D. E. Corlett, H. F. Cowper Johnson, F. S. Cozens, J. L. Crammer, R. C. Curry, D. M. Davis, A. E. L. de Thierry, J. A. Dew-Jones, D. G. Dingle, H. Eaton, B. I. F. Eminson, J. P. English, J. A. Fiddian, J. R. T. Finlayson, M. V. J. Fitzgerald, N. W. Fogg, M. G. A. Forrest, R. Gardner, R. H. Gibbon, J. T. Gibbs, D. N. M. Goodson, B. G. Gradwell, R. K. Greenwood, A. M. Hammerton-Fraser, J. W. Harding, W. B. Hepburn, M. Heslop, F. Heyes, G. H. Hilton, C. B. Hobbs, H. J. Hodgson, M. Horowitz, R. Hutchinson, P. O. Jones, Mrs. E. S. Kerr, R. J. Knight, R. J. W. Lambert, J. E. Lennard-Jones, I. S. Lister, J. S. Lloyd, S. P. Lock, D. E. Longden, F. McP. Low, J. A. McEwan, C. M. McGregor, N. D. Mackichan, Mrs. J. F. Mackworth, A. F. Madge, I. M. J. Mair, G. F. Mayall, G. Meachim, R. A. Miley J. S. Miller, E. J. Mitchell, D. G. Montefiore, R. L. Morgan, M. H. P. Muir, J. C. Newell Price, K. R. Oliver, E. M. Orton, I. Oswald, J. Paterson, C. J. Pattinson, M. J. W. Penn, J. G. Pritchard, D. K. Robinson, Mrs. L. M. Rochelle, R. A. Roxburgh, H. Schlossmann, P. Sleight, I. M. P. Smeed, P. T. Smith, J. K. Snell, E. J. Stacey, G. P. Starck, C. J. Stephens, J. L. Stevens, I. A. Stewart, L. J. Stretton, S. G. Sutton, I. G. Tait, J. L. Tasker, H. T. Thomas, C. P. D. Tong,

R. M. Turner, T. M. Vanier, G. P. Vaughan, J. D. S. Vernon, P. C. Vivian, A. E. Wagstaff, E. H. M. Walker, R. K. Waterhouse, J. H. H. Webster, P. Wellings, F. L. Whalley, E. D. Williams, G. C. E. Wilson, J. O. Winter, N. W. H. Young.

UNIVERSITY OF ST. ANDREWS

The following candidates have been approved at the examinations indicated:

M.D.—R. A. B. Rorie.

FINAL M.B., CH.B.—K. Davies, C. Gunn, Julia C. Izzard, R. M. Kelly, Eileen M. Key, E. D. McEwan, H. R. Simpson, Elizabeth M. Stephen.

UNIVERSITY OF GLASGOW

In Congregation on December 19, 1952, the following medical degrees and diplomas were conferred:

CH.M.—R. B. Wright (with high commendation).

PH.D.—T. W. Wikramanayake, M.B., B.S.

M.B., CH.B.—G. J. Addis, J. C. Baird, A. T. Bowie, Mavis J. Bowyer, Z. D. Brodie-Mends, D. B. Campbell, G. R. Carroll, N. Chazan, Joan A. Cree, G. L. W. Cunningham, A. F. Davidson, J. McK. Davie, J. A. Docherty, A. Dodds, T. E. S. Fergusson, Morag B. P. Fernie, H. F. Frame, D. A. S. Fraser, Annice H. G. Gillespie, A. K. Gillies, Joyce E. J. Hassan, H. Hatrick, T. Hetherington, W. E. I. Healy, D. E. Jardine, J. S. Kennedy (with commendation), W. McG. Kirk, S. J. Lepper, Mrs. David G. Lindsay (née Mactaggart), Eileen M. MacColl, F. McCormack, A. Macdonald, Jean Y. MacDougall, A. G. R. McFadyen, J. McGlone, S. W. McGowan, Barbara S. Maclean, D. Macmillan, T. E. Macnamara, A. S. MacPhail, I. Macpherson, F. McRae, R. Meldrum, C. M. Morton, R. O. Nkeaka, A. R. Pate, R. Reid, H. G. Robinson, E. D. T. Ross, G. Ross, G. B. Ross, Joyce W. Russell, J. H. Shearer, W. M. Shovlin, A. Sinclair, J. D. Stevenson, Sheena G. MacD. Stuart, J. Sutherland, A. S. Taylor, A. S. Thomson, Dorothy E. Walby, Ethna A. Walshe, Doris M. White, J. G. Young, Jean McI. Young.

D.P.H.—J. McGovern.

UNIVERSITY OF EDINBURGH

At a Graduation Ceremonial on December 19, 1952, the following degrees and diplomas were conferred:

M.D.—*A. Cameron.

PH.D.—*In the Faculty of Medicine:* *S. W. Bibile.

M.B., CH.B.—F. E. Adderley, Jean M. Bell, G. Beveridge, Jean Bheenick, D. A. Brown, J. D. Cormack, J. T. Dixon, A. G. Fraser, A. H. French, A. K. Fulton, Alice B. E. Gray, Ann S. Haig, R. W. Hall, D. L. N. Haultain, P. A. Hood, Isabel J. Huggan, A. D. L. Hunter, J. L. Hunter, R. D. Jones, P. L. Kennedy, D. S. Lyon, R. B. Macpherson, R. S. Maini, D. J. B. Makins, N. E. L. Margetson, A. R. Mather, Q. S. Moore, Elizabeth M. Munro, W. S. Munro, G. M. Ogilvie, T. G. Paterson, W. B. Paterson, J. M. Robertson, I. Shaw, Catherine J. Tulloch, R. P. M. Urquhart, B. Willis, F. F. Wilson, W. M. Wilson, Elizabeth J. Q. Wood, J. C. Yellowley.

B.SC.—*In the Department of Pure Science:* *J. S. Milne, M.B., Ch.B.

DIPLOMA IN MEDICAL RADIODIAGNOSIS.—F. G. T. Beetham, D. A. E. Dewar, R. K. McFarlane, D. A. R. Ritchie.

DIPLOMA IN MEDICAL RADIOTHERAPY.—A. R. Lyons, K. S. Preston, J. B. McEwan.

The following scholarships, etc., were awarded in the Faculty of Medicine: *Crichton Research Scholarship,* Dr. Evelyn A. C. Skinner. *Hastilow Research Scholarship in Rheumatism,* Dr. W. R. M. Alexander. *Andrew Graham Ritchie Bursaries,* A. W. McKenzie, J. T. Wilson, E. N. C. Milne. *Coldstream Memorial Medical Missionary Bursary,* W. A. M. Cutting. *Neil Arnott Prize in Experimental Physics,* W. J. Irvine. *Russell-Crighton Prize,* J. Syme.

*In absentia.

UNIVERSITY OF DUBLIN

SCHOOL OF PHYSIC, TRINITY COLLEGE

The following candidates have been approved at the examinations indicated:

M.D.—B. P. P. Berney, D. W. Montgomery, S. Smullen, M. S. Strong.

FINAL M.B., B.CH., B.A.O.—[1]Betty E. Wallace, K. W. H. B. McAlpine.

FINAL M.B., B.CH.—M. C. Latham, Christina Davison, J. R. Polson, Mavis Sayers.

FINAL M.B.—R. G. B. Williamson, J. E. Latham, W. B. C. Fagan, J. C. H. Haythornthwaite, W. J. C. Stevenson, P. E. A. Kalunta-Onyejekwe.

FINAL M.B., B.A.O.—Joyce M. Fullerton, G. R. L. Lyons, Margaret E. E. Graham.

FINAL B.CH.—[1]Leila L. Dyer, [2]Anna M. K. Little, [2]R. Indar, J. J. G. Kneafsey, O. R. McCarthy, B. A. Montgomery, Margaret E. Charleton, D. D. Chesney, Sheila King, Adebola Bailey, R. M. Bowman, H. W. Bridge, M. M. Berber, Ethel G. Agnew, M. A. McVicker, H. M. O. Brown, Marie T. Hyde, G. M. Irvine, Ruth A. Helsby.

FINAL B.CH., B.A.O.—H. Hardman.

FINAL B.A.O.—S. Baigel, Sarah B. Schwartzberg, C. A. H. Trench, M. M. Haniff, F. Fine.

The following medical degrees were conferred on December 4:

M.D.—D. W. Montgomery.

M.B., B.CH., B.A.O.—M. M. Berber, R. M. Bowman, H. W. Bridge, H. M. O. Brown, Margaret E. Charleton, D. D. Chesney, Leila L. Dyer, W. B. C. Fagan, J. C. H. Haythornthwaite, Ruth A. Helsby, Marie T. Hyde, R. Indar, G. M. Irvine, Sheila King, J. J. G. Kneafsey, M. C. Latham, Anna M. K. Little, K. W. H. B. McAlpine, O. R. McCarthy, M. A. McVicker, B. A. Montgomery, J. R. Polson, Mavis Sayers, Sarah B. Schwartzberg, C. A. H. Trench, Betty E. Wallace, R. G. B. Williamson.

[1]Second-class honours in medicine. [2]Second-class honours in surgery.

UNIVERSITY OF DURHAM

In Congregation on December 19, 1952, the following medical degrees were conferred:

M.B., B.S.—B. T. M. Aboko-Cole, Kathleen D. Blackett, Q. A. Bynoe, R. Chambers, Dorothy H. Cooper, Ann E. Crabbe, E. P. Daniels, C. C. Day, G. MacA. Dowson, K. W. H. Fenwick, F. M. Fyfe, Edith P. Gilbertson, J. C. Gilbertson, J. C. Gilmour, H. E. Harper, A. G. Larson, Moira Lickley, C. R. Markham, Jean A. Rosenstein, I. M. Telfer, A. Turner, I. A. R. Watson, A. White.

UNIVERSITY OF LONDON

The Sanderson-Wells Senior Prize has been awarded to Dr. Anthony Michael Dawson (Charing Cross Hospital Medical School) and the Junior Prize to Mr. Jeffrey Eaton Cullis (St. Thomas's Hospital Medical School); the A. H. Bygott Scholarship to Dr. Antony John Essex-Cater; the Sir William Dunn Scholarship to Mr. Derek John Goodman (St. Mary's Hospital Medical School); and a University Studentship in Physiology to Mr. Alexander Angus Buchanan (St. Mary's Hospital Medical School).

Professor B. W. Windeyer has been appointed delegate of the University at the Seventh International Congress of Radiology to be held at Copenhagen from July 19 to 25, 1953, and Professor J. M. Mackintosh delegate at the Health Congress of the Royal Sanitary Institute to be held at Hastings from April 28 to May 1, 1953.

The following have been appointed or nominated for appointment or reappointment as representatives of the University on the governing bodies indicated in parentheses: Dr. Katharine G. Lloyd-Williams (Cheltenham Ladies' College); Dr. A. R. Kagan (West Kensington School); Mr. St. G. B. Delisle Gray (Cranbrook and Morpeth Schools); Dr. Stephanie M. Leese (John Harvard, Nelson and Borough Paragon Schools).

The following have been recognized as teachers of the University in the subjects indicated in parentheses: *Institute of Neurology,* Dr. William Blackwood (Pathology (Neurohistology)). *Institute of Dental Surgery,* Mr. W. G. Cross, M.S. (Dental Surgery (Periodontology)). *Lister Institute of Preventive Medicine,* Dr. A. A. Miles (Pathology and Bacteriology). *Chelsea Polytechnic,* Dr. Mary F. Rockett (Physiology and Pharmacology).

The list of institutions recognized for the M.B., B.S. degrees for external students has been amended by the deletion of the present entry under Oxford and by the substitution of the following: Oxford: The University Medical School with the United Oxford Hospitals; the Wingfield-Morris Hospital (for Orthopaedics only).

The John Marshall Fellowship in Surgical Pathology of the value of £700 a year, for two years in the first instance, and which is tenable at the University College Hospital Medical School, has been awarded to Arnold Lawrence Levene, M.B., B.S.

J. A. Pedler, M.R.C.S., L.R.C.P., B.D.S., L.D.S., has been approved at the examination for the degree of M.D.S.

The title of Professor of Dental Surgery has been conferred on Frank Clare Wilkinson, M.D., D.D.Sc., F.D.S., Director of the Institute of Dental Surgery, British Postgraduate Medical Federation.

The following candidates have been approved at the examination indicated:

M.S.—*Branch I (Surgery):* C. Brun, W. D. Corfield, D. H. Jenkins, P. F. Philip, M. C. T. Reilly, H. H. Renyard, J. J. Shipman, K. W. Wilkinson.

THIRD M.B., B.S.—[1 4] V. Ainsworth, [1 4 5 6 7] A. J. Bowdler (University Medal), [1 4] J. Brown, [1 7] J. Ernsting, [1 7] Kathleen M. Huntington, [1 6] W. J. Jones, [1 6] Violet H. Meyerstein, [1 5] Doreen M. Nutbourne, [1 2 4] J. I. S. Robertson, [1 3] R. J. Shephard, [1 6] R. P. Wise, Lilian E. Ackermann, E. D. B. Allen, L. G. Andrews, J. M. Armitt, J. A. Armstrong, K. M. Backhouse, A. H. W. Bain, D. T. C. Barber, Joyce L. Barber, N. H. Barley, D. W. Barnes, I. R. Beale, A. Betts, J. F. Bird, Joan M. Bishop, S. W. Bissell, Joy S. Blight, R. Blight, M. Bloch, E. A. Boulter, S. Bourne, J. G. Bradford, R. F. Braithwaite, Louise A. Brandram, J. H. Breed, E. W. Brentnall, C. P. B. Brook, M. D. Brydson, Mavis Buck, J. Caplan, Margaret S. Cavendish, E. J. Chalker, P. J. C. Chapman, H. P. Charles, Lisbeth S. Chazan, D. A. Chiswell, R. H. Clark, L. J. Clark-Wilson, Elizabeth P. Cloake, T. J. Clyne, Evelyn Coates, P. J. Cook, C. W. Coole, A. F. C. Coppin, M. P. M. Coupland, J. C. Coury, B. J. Cremin P. N. Cretney, D. M. Cuthbert, Pauline M. Daniels, A. Davies, P. E. Davies, L. M. Davis, E. C. Dawson, V. E. Dean, W. M. A. Derla, P. A. de Raeve, Margaret M. Derrington, J. E. Dessart, R. Dewar, Sheila Dewhurst, D. M. Digues La Touche, P. Dilnot, T. L. Dormandy, A. E. Dossetor, R. E. Dreaper, Sylvia I. Duckworth, T. Duffy, C. J. E. Dusseau, J. J. H. Eastwood, C. J. R. Elliott, Isabel K. M. Elmslie, L. L. Emery, C. A. S. Emmett, Anne C. Evans, Gwenda W. Evans, M. D. C. Evans, F. W. Fairhead, M. Fanning, P. M. Forster, A. I. Fram, J. L. Fraser, A. G. E. Fung-a-Fatt, D. R. Gamble, F. Gaskell, H. R. George, P. R. Gilbert, G. A. Ll. G. Glazebrook, G. D. Glennie, R. M. H. Gompertz, Stephanie J. Goodin, A. W. G. Goolden, D. E. Granger, C. M. Grant, G. C. Grassby, D. K. Gray, D. J. Green, Elizabeth A. Grimditch, M. J. Gryspeerdt, P. J. Guly, Christine E. E. Hammond, E. H. L. Harries, Joyce Harrison, K. A. Harwood, J. R. K. Henry, E. R. Herst, J. P. N. Hicks, F. A. Hill, W. Hindle, J. Hirsh, C. B. Hodes, D. Hooker, C. J. Hopkins, B. J. Houghton, C. K. Hudson, K. R. Hughes, J. P. Hurrell, V. H. Hutchinson, C. F. Hutchison, P. L. Huxley-Williams, Anne L. Jacobs, W. H. James, D. G. W. Jenkins, P. B. Jensen, Gwyneth M. V. Johns, H. S. Jones, H. D. Jones, E. E. F. Keal, R. J. Kennedy, Patricia M. Kenney, D. J. Kidd, D. W. King, D. W. G. King, J. F. King, Iris M. Krass, Margaret A. Lakeman, A. N. Lamplugh, S. P. Lapage, A. V. Large, J. D. Le Bouvier, M. J. F. Lee, Monica C. Lewin, Rachael B. J. Lewis, A. B. Lodge, E. C. Long, E. E. Low, J. G. Lymbery, Judith Lyne, Kathleen E. Mackinnon, Diana O. McKnight, P. MacMahon, W. E. H. Maeckelberghe, Nancy S. Maguire, R. D. Mann, Stella M. Manners, J. Manuel, Philippa M. Martin, J. F. A. Maskell, R. A. Matthews, T. J. K. Merritt, I. Mohammed, E. T. Mold, Ann G. Morgan, D. C. Morrell, B. A. Morrison, I. G. Mowat, E. A. Murray, R. G. Newberry, E. W. P. Noall, P. R. Norris, M. D. O'Riordan, W. G. Owen, F. M. Owers, A. R. W. Page, J. R. Palmer, F. I. Partington, P. F. Peeks, Naomi I. Penfold, R. J. V. Peter, G. I. Phillips, R. V. Pierson, A. J. Platts, A. P. Pritchard, D. S. Ractliffe, F. C. Ramsey, J. Randall, H. F. Reichenfeld, C. E. Rhodes, Dorothy M. Roberts, J. K. Roberts, T. E. Roberts, M. P. Robinson, Barbara E. C. Robson, R. S. B. Rogers, S. K. Rogers, Jean L. Romanes, M. E. Rope, E. M. Rosser, P. E. H. Rutter, Elizabeth A. Ryan, J. F. Ryan, D. H. Ryde, M. G. Selson, J. E. Shahmoon, F. M. Shattock, D. B. Shaw, B. J. Sheerin, Mary S. Shenai, Elizabeth L. Siddle, G. E. Singer, F. B. Singleton, June T. Slatter, W. D. A. Smith, A. Spencer, S. P. Spiers, T. J. Stanton, J. L. Stevenson, Kathleen M. Stevenson, M. F. Sturridge, R. V. Sutton, D. A. Symers, Elizabeth J. Taylor, M. G. Taylor, A. J. Third, Patricia M. Thompson, Lorna G. Tolchard, Maureen A. Tudor, P. J. van den Brul, A. A. A. C. G. van der Bracht, A. G. Villeneau, D. Watkins, G. C. Watson, Joyce D. Waye, R. A. J. Webb, J. S. Wesson, Janet M. White, J. H. White, Gladys M. H. Williams, J. H. Williams, Eileen M. Williamson, J. B. Williamson, R. Wilton, Mary A. Winser, Sylvia H. Wollner, A. R. Worters, A. P. Wright, D. A. Wright, D. E. Wright, F. A. Yates.

ACADEMIC POSTGRADUATE DIPLOMA IN MEDICAL RADIOLOGY (DIAGNOSIS).—J. McN. Lockie, E. Mercieca.

ACADEMIC POSTGRADUATE DIPLOMA IN PSYCHOLOGICAL MEDICINE.—E. F. Carr. *Part I:* M. G. Jayasundera, Mary M. McQuade, M. Pines.

[1]With honours. [2]Distinguished in pathology. [3]Distinguished in hygiene and forensic medicine. [4]Distinguished in medicine. [5]Distinguished in applied pharmacology and therapeutics. [6]Distinguished in surgery. [7]Distinguished in obstetrics and gynaecology.

UNIVERSITY OF MANCHESTER

The following candidates have been approved at the examination indicated:

FINAL M.B., CH.B.—Rachel M. Arnold, J. K. H. Benson, Enid M. Carpenter, V. E. Chase, J. Clarke, Meryl P. Clynes, Helen R. Ellis, Phyllis E. Evans, R. H. Finnie, D. J. Fitzmaurice, D. H. Flynn, H. Greenberg, H. H. Gunson, L. D. Harris, Heather J. Jaffe, K. Kay, E. J. B. McArthur, D. Milner, Myfanwy C. R. Morris, J. H. Muende, A. S. Nicholas, B. Quinn, G. W. Savatard, S. W. Singleton, J. F. Tomlinson, Margaret A. Wignall, A. Wilson, A. H. Woodhall.

UNIVERSITY OF WALES

The Council of the Welsh National School of Medicine has appointed Hubert John Whiteley, M.D., to the post of Senior Lecturer in the Department of Pathology and Bacteriology of the School. Dr. Whiteley is at present Lecturer in Pathology in the University of Sheffield.

In Congregation on December 19, 1952, the following medical degrees were conferred:

M.B., B.CH.—R. F. Addy, Edna Brennan, L. V. Chubb, T. Gunner, Jeanne G. Roper, A. O. Thomas, J. E. Watkin, D. J. Ll. Williams.

UNIVERSITY OF BIRMINGHAM

Professor W. Melville Arnott has been appointed Deputy President of the first World Conference on Medical Education to be held in London in August, 1953.

Professor A. P. Thomson has been appointed representative of the University on the Inter-University Council for Higher Education in the Colonies.

A. B. MacGregor, M.D., F.D.S., has been appointed John Humphreys Professor of Dental Surgery in the University from October 1, 1953.

Drs. G. A. Dalton and J. N. Ormrod have been appointed temporary Assistant Lecturers in Anatomy from October 1, 1952.

Dr. Stanley A. Way has been invited to give the Ingleby Lectures in 1953.

UNIVERSITY OF LIVERPOOL

The following candidates have been approved at the examinations indicated:

M.D.—H. G. Frank, D. R. W. Haddock, A. Jolleys, R. S. Jones, P. P. Newman.

M.CH.ORTH.—C. J. R. Conacher, W. C. Lawrence, M. Lunz, R. Pagé, A. Rahim, E. H. Strach, J. G. Stubbs, K. J. M. Watson.

FINAL M.B., CH.B.—*Part IV:* Gwerfyl Alun Roberts, Ursula M. Anderson, S. R. Bennett, Margaret T. Brash, J. R. Davison, C. Gregory, L. A. Guile, J. Hosker, R. D. Jones, J. B. Kenyon, I. McGilvray, I. Miller, L. A. Nettleton, W. G. W. Parry, T. D. Thomas, N. Wignall, Margaret M. Williams, J. D. Wright, Madge Yates. *Passed in Separate Subjects:* Eileen M. Howarth (Medicine and Surgery). *Part III:* Margaret J. Alltree, S. J. Alltree, Cicely G. Brenner, R. H. Cranmer-Gordon, L. M. Ellis, H. Halsall, G. M. Makhlouf, D. R. Morris, L. H. le Riche, J. S. Robinson, R. T. Shepherd, P. J. J. Wren.

DIPLOMA IN PUBLIC HEALTH.—R. Muscat. *Part I:* Alice T. McGlinchey, J. McHugh, A. N. Pickles, W. A. Pollitt, L. G. Poole, Z. D. A. Sheikh, Sheila M. Towers.

CERTIFICATE IN PUBLIC HEALTH.—Catherine M. Connolly.

DIPLOMA IN RADIOLOGY (RADIOTHERAPY).—R. V. Samapurnavanitya.

UNIVERSITY OF LEEDS

The title of Dr. T. A. Divine, at present Senior Administrative Officer of the School of Medicine, will be in future Sub-Dean of the Faculty of Medicine, and that of the Administrative Assistant to the Senior Administrative Officer will be Secretary to the School of Medicine.

Dr. R. G. Paley has been appointed Research Fellow in Medicine for two years from October 1, 1952.

Dr. L. L. Dmochowski, Reader in Cancer Research, has been invited to become Visiting Associate Professor of Microbiology at the College of Physicians and Surgeons, Columbia University.

Dr. G. W. Theobald has been appointed Honorary Lecturer in the Department of Obstetrics and Gynaecology and Dr. Max Hamilton Senior Lecturer in Psychiatry.

Dr. B. D. Wyke has resigned from his lectureship in the Department of Physiology, as from January 31.

UNIVERSITY OF SHEFFIELD

Dr. D. R. Wood has been appointed Senior Lecturer in Pharmacology and Dr. T. M. Abbas Lecturer in Obstetrics and Gynaecology.

The Council of the University has received the following resignations: Dr. S. Oleesky, Lecturer in Therapeutics, on his appointment as Consultant Physician at Crumpsall Hospital, and Dr. H. J. Whiteley, Lecturer in Pathology, on his appointment as Senior Lecturer in Pathology at the Welsh National School of Medicine.

In Congregation on December 16 the following medical degrees were conferred:

M.D.—J. D. Hopewell, C. W. Lawson.

M.B., CH.B.—D. P. Adams, R. Cartledge, Margaret B. Dun, J. P. Green, N. B. Harris, A. D. Heath, J. K. Mensah, B. A. Thompson, G. N. W. Tilsley.

UNIVERSITY OF BRISTOL

The following candidates have been approved at the examinations indicated:

FINAL M.B., CH.B.—K. J. Barker, Marjorie E. Barton, N. G. Blackwell, A. R. Bollen, Ann E. Booker, J. C. Causton, Anne W. Christie, B. Ginsberg, A. Moore, I. W. Palmer, M. N. Prichard, P. H. Sherwood, H. S. Smith, D. W. G. Stone, J. E. Tovey. *In Group I completing the examination:* Jean M. Claremont, H. A. P. Walker. *In Group I only:* Elizabeth M. James.

ROYAL COLLEGE OF SURGEONS OF ENGLAND

At a meeting of the Council of the College on December 11, with Sir Cecil Wakeley, President, in the chair, a Moynihan Lectureship was awarded to Dr. Richard B. Cattell, of the Lahey Clinic, Boston; Mr. G. T. Hankey was appointed Charles Tomes Lecturer for 1953; Professor H. F. Humphreys, Vice-Chancellor of the University of Birmingham, was appointed to give the second Webb-Johnson Lecture in May, 1954; and the Begley Prize was presented to J. H. Breed (Westminster Hospital).

Diplomas of Fellowship were granted to the following successful candidates: J. S. Karanwal, C. H. J. Rey, E. H. L. Cook, J. R. G. Edwards, J. N. Aston, F. Glassow, E. A. Malkin, L. Turgill, P. S. Jaikaran, D. G. Maurice, R. J. Moolan-Feroze, Helen M. Mellor, P. R. French, M. F. Hunt, G. L. Bourne, T. H. Morgan, L. A. J. Evans, M. H. Stroud, W. R. Denny, P. G. Bevan, Irene M. S. Cade, A. J. Harrold, C. Havard, D. J. Oakland, R. A. Payne, R. L. Grynoch, D. W. T. Roberts, H. R. Ker, Joyce M. Daws, C. Nagrath, B. P. Chatterjee, A. K. Gupta, H. E. Marsden, H. I. LeBrun, P. D. Blaxland, J. C. FitzHerbert, D. R. Ll. Hart, M. P. Pai, H. M. Shaw, E. L. G. Beavis, L. Blumberg, W. B. Law, Cicely Pepler, R. R. Spencer, B. J. S. Grogono, A. St. F. Henley, G. G. McCloskey, D. H. McMillan, M. A. Majid, A. G. Moffoot, R. K. Newing, L. H. Pimm, N. L. Pramanick, G. E. Schofield, G. K. Thomas, M. A. Birnstingl, J. E. Blundell, D. J. Crockett, Kathleen A. M. Frith, N. T. Hamilton, T. A. H. Hurrell, R. O. Parnis, B. E. Pierard, T. Savage, M. H. G. K. Siriwardena, J. B. L. Taylor, J. G. Toakley, D. J. Tuffley, S. E. Wijetilaka, G. G. Wyllie, R. Alhadeff, Y. Cohen, J. L. Connell, W. T. Irvine, D. G. Lane, H. K. McComb, B. V. McEvedy, P. D. O'Donoghue, G. B. Ong, A. D. Roy, G. Wilson, A. F. Grant, G. M. Linehan, T. P. Nash, N. A. Shah, J. H. Shoesmith, S. R. Young, J. S. Brown, W. D. Friend, V. M. Kini, R. M. Mitchell, J. B. Self, R. T. Turner Warwick.

A Diploma of Membership was granted to J. A. McHardy (Guy's).

Hospitals were recognized under paragraph 23 of the F.R.C.S. regulations as follows: Brighton General Hospital, resident surgical officer for one year; house-surgeon (general surgery) for six months. Doncaster Royal Infirmary (*decennial revision*), surgical registrar for six months; two house-surgeons for six months. Wolverhampton and Midland Counties Eye Infirmary, (*additional recognition*), *Paragraph 23b*, senior registrar. Royal Surrey County Hospital, Guildford, *Paragraph 23c*, registrar and house-surgeon to ear, nose, and throat department. Chester Royal Infirmary, *Paragraph 23c*, registrar to ear, nose, and throat department. South Devon and East Cornwall Hospital, Plymouth, *Paragraph 23c*, registrar to ear, nose, and throat department. Ingham Infirmary, South Shields (*additional recognition*), two house-surgeons for six months (until December 31, 1955). Perth Royal Infirmary, two house-surgeons for six months; registrar for one year. Royal Hospital, Wolverhampton (*decennial revision*), resident surgical officer for one year: *for six months*, three house-surgeons (general surgery); senior house officer (fracture and orthopaedic unit); house-surgeon (fracture and orthopaedic unit); casualty registrar. Hospital of St. Cross, Rugby, *for six months*, resident surgical officer; house-surgeon (general); orthopaedic registrar. North Riding Infirmary, Middlesbrough, *Paragraph 23b*, ophthalmic registrar; ophthalmic house-surgeon. Bridge of Earn Hospital, *for six months*, three house-surgeons (general surgery); three orthopaedic house-surgeons; three orthopaedic senior house officers; two orthopaedic registrars.

ROYAL FACULTY OF PHYSICIANS AND SURGEONS OF GLASGOW

At a meeting of the Faculty, held on December 1, with Dr. Andrew Allison, President, in the chair, the following were admitted Fellows of Faculty: W. M. Dennison, J. C. Dick, B. Isaacs, L. Schamroth, Marjorie B. Snodgrass, D. G. C. Whyte.

ROYAL COLLEGE OF PHYSICIANS OF IRELAND

The following were admitted to the Membership of the College on November 7: D. F. Cantwell, J. A. C. de Silva, J. C. McGoldrick, K. M. Hickey, R. J. Marshall, E. M. A. McElligott, G. E. Peters, A. L. Wells.

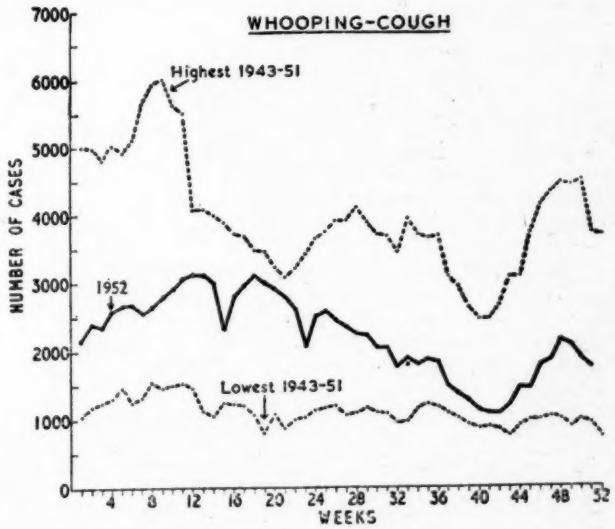
Vital Statistics

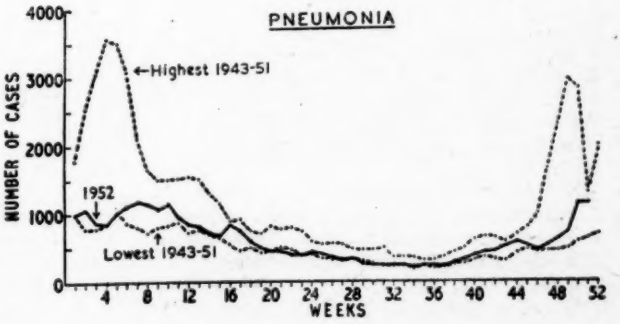
Week Ending December 27

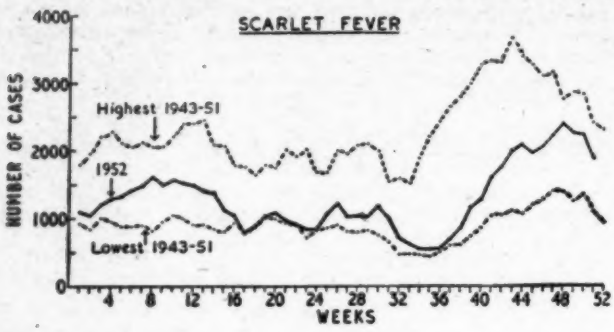
The notifications of infectious diseases in England and Wales during the week included: scarlet fever 1,358, whooping-cough 1,117, diphtheria 26, measles 14,189, acute poliomyelitis 33, dysentery 184, paratyphoid fever 3, and typhoid fever 2.

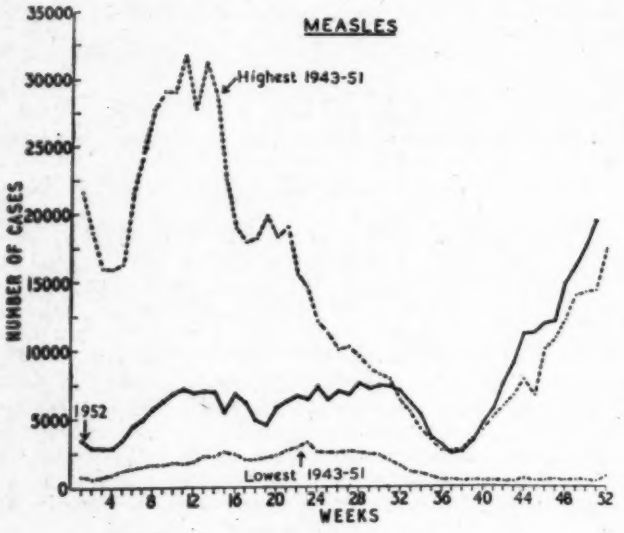
Graphs of Infectious Diseases

The graphs below show the uncorrected numbers of cases of certain diseases notified weekly in England and Wales. Highest and lowest figures reported in each week during the nine years 1943–51 are shown thus - - - - - - -, the figures for 1952 thus ———. Except for the curves showing notifications in 1952, the graphs were prepared at the Department of Medical Statistics and Epidemiology, London School of Hygiene and Tropical Medicine.









Infectious Diseases

Measles, with a rise of 1,809 in the number of notifications, from 17,742 to 19,551, was the only infectious disease with an increased incidence during the week ending December 20, 1952, in England and Wales. There were decreases of 348 for scarlet fever, from 2,229 to 1,881, and 117 for whooping-cough, from 1,865 to 1,748.

The increase in the incidence of measles was mainly in London and the Home Counties; the largest rises were Essex 515 (from 1,812 to 2,327), Middlesex 406 (923 to 1,329), London 393 (1,404 to 1,797), Kent 232 (949 to 1,181), and Warwickshire 212 (903 to 1,115). The largest falls in the number of notifications of measles were 124 in Leicestershire (566 to 442) and 90 in Durham (530 to 440). The decrease in the incidence of scarlet fever was general throughout the country, the only large fall being 92 in Yorkshire West Riding, where the notifications decreased from 275 to 183. Only small fluctuations were recorded in the local trends of whooping-cough. The number of notifications of diphtheria (23) was the same as in the preceding week. The chief features of the local returns of diphtheria were a fall from 4 to 0 in Devonshire and a rise from 1 to 7 in Warwickshire, where all the cases were notified in Birmingham C.B.

47 cases of acute poliomyelitis were notified during the week; these were 6 fewer for paralytic and 5 fewer for non-paralytic cases than in the preceding week. The largest returns were Yorkshire West Riding 9 (Sheffield C.B. 3), London 6, Middlesex 5, Lancashire 5.

327 cases of dysentery were notified—one fewer than in the preceding week. The chief centres of infection were Lancashire 59 (Bolton C.B. 18, Clitheroe R.D. 14, Liverpool C.B. 13), Surrey 54 (Carshalton U.D. 46), Lincolnshire 49 (Grimsby C.B. 46), Yorkshire West Riding 47 (Barnsley C.B. 30), London 34, Kent 30 (Penge U.D. 26).

INFECTIOUS DISEASES AND VITAL STATISTICS

Summary for British Isles for week ending December 20 (No. 51) and corresponding week 1951.

Figures of *cases* are for the countries shown and London administrative county. Figures of *deaths* and *births* are for the 160 great towns in England and Wales (London included), London administrative county, the 17 principal towns in Scotland, the 10 principal towns in Northern Ireland, and the 14 principal towns in Eire.

A blank space denotes disease not notifiable or no return available.

The table is based on information supplied by the Registrars-General of England and Wales, Scotland, N. Ireland, and Eire, the Ministry of Health and Local Government of N. Ireland, and the Department of Health of Eire.

CASES in Countries and London	1952					1951				
	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire
Diphtheria	23	3	6	0		54	5	11	1	1
Dysentery	327	34	77	4	4	422	29	148	3	1
Encephalitis, acute	7	0	0	0		1	0	0	0	
Enteric fever:										
Typhoid	5	0	0	0		6	0	0	0	1
Paratyphoid	1	0	1 (B)	0		17	0	5 (B)	5	1 (B)
Food-poisoning	58	8		0		182	10		1	
Infective enteritis or diarrhoea under 2 years				6	14				16	14
Measles*	19,551	1797	181	23	40	2,321	28	134	159	85
Meningococcal infection	38	0	16	2	1	30	2	12	1	4
Ophthalmia neonatorum	29	2	5	0		27	1	10	0	
Pneumonia†	1,141	99	413	5	4	681	57	238	3	1
Poliomyelitis, acute:										
Paralytic	35	3	3	2	2	22	0	9	2	0
Non-paralytic	12	3				6	1			
Puerperal fever§	160	29	12	2		203	22	12	1	2
Scarlet fever	1,881	110	320	48	60	1,552	112	292	21	44
Tuberculosis:										
Respiratory			147	15				126	27	
Non-respiratory			19	3				31	10	
Whooping-cough	1,748	90	102	57	65	1,819	63	262	74	86

DEATHS in Great Towns	1952					1951				
	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire	Eng. & Wales	Lond.	Scot.	N. Ire.	Eire
Diphtheria	1	0	0	0	0	*0*	0	*0*	0	0
Dysentery	0	0		0		–	0		0	
Encephalitis, acute		0					0			0
Enteric fever	0	0	0	0		*0*	0	*0*	0	
Infective enteritis or diarrhoea under 2 years	18	4	3	0	0	*21*		*2*	0	2
Influenza	45	9	3	0	0	*13*	3	*0*	1	0
Measles		0	0	0	0		0	*1*	0	1
Meningococcal infection		3	0				0	*0*		
Pneumonia	515	125		16	9	*273*	55		11	9
Poliomyelitis, acute	6	3			0	*2*	0			0
Scarlet fever		0	0	0	0		0	*0*	0	0
Tuberculosis:										
Respiratory	160	37	14	5	4	*139*	26	*17*	3	7
Non-respiratory		2	3	0	1		1	*3*	0	3
Whooping-cough	1	0	2	0	0	*1*	0	*0*	0	0
Deaths 0–1 year	256	34	42	9	12	*257*	29	*31*	15	22
Deaths (excluding stillbirths)	7,679	1523	746	143	125	*5,685*	951	*660*	135	183
LIVE BIRTHS	6,764	1045	769	197	277	*6,405*	1050	*787*	267	320
STILLBIRTHS	168	13	25			*192*	37	*17*		

* Measles not notifiable in Scotland, whence returns are approximate.
† Includes primary and influenzal pneumonia.
§ Includes puerperal pyrexia.

Medical News

College of General Practice.—Dr. G. F. Abercrombie, a London general practitioner and former president of both the Hampstead Medical Society and the General Practice Section of the Royal Society of Medicine, has been elected chairman of the foundation council of the College of General Practice. The deputy chairman is Dr. F. M. Rose, and the honorary secretary Dr. John Hunt.

British Association.—Sir Edward Appleton, F.R.S., Vice-Chancellor of Edinburgh University, has been appointed President of the British Association for the Advancement of Science, in succession to Professor A. V. Hill, F.R.S.

Local Medical Societies.—Meetings held last month included the following: *Manchester Medical Society.*—On December 3 Sir Geoffrey Jefferson, F.R.S., read a paper to the Section of Medicine on "The varying behaviour of pituitary adenomata," and on December 9 Dr. R. W. Fairbrother addressed the Section of Surgery on "Antibiotics." *Chelsea Clinical Society.*—On December 16 there was a discussion on "Juvenile delinquency," opened by Mr. Basil Henriques, J.P., and Dr. William Moodie. *Devon and Exeter Medico-Chirurgical Society.*—On December 18 Dr. W. A. Lister spoke on "Common out-patient problems." *North of England Obstetrical and Gynaecological Society.*—On December 19 Mr. P. Malpas delivered his presidential address on "The nature of biological causality."

Association of Plastic Surgeons.—At the annual general meeting of the British Association of Plastic Surgeons, held in London last month, Mr. J. N. Barron was elected president, Mr. D. N. Matthews vice-president, Mr. R. P. Osborne hon. treasurer, and Mr. J. P. Reidy hon. secretary.

Refresher Course Book.—The first impression of the book of "Refresher Course" articles has now sold out. A second impression is being prepared and is expected to be available by the end of this month. Orders may be placed now with the Publishing Manager, B.M.A. House, Tavistock Square, W.C.1, for both the Refresher Course Book (25s.) and for the second book of "Any Questions?" (7s. 6d.; postage 6d.).

Metropolitan E.N.T. Hospital.—The medical staff of the Metropolitan Ear, Nose, and Throat Hospital have now agreed to the moving of the hospital's in-patient department to St. Mary Abbots Hospital, Kensington (see *Journal*, December 6, p. 1267). Operating has already begun there.

Dr. Gow's Will.—St. Bartholomew's Medical College has declined the offer of the late Dr. A. E. Gow's body for dissection, or for display in the hospital museum after cremation "so that the students may see the volume and weight of residue from an adult." Dr. Gow was consulting physician to St. Bartholomew's Hospital and honorary physician to the Household of the Duchess of Kent (*Journal*, October 4, 1952, p. 781).

Electrocution of Unwanted Animals.—The Universities Federation for Animal Welfare, of which Professor Edward Hindle, F.R.S., is president, has been sponsoring research on methods of electrocuting animals. One of the objects of this work is to ensure that pain is not inflicted. A duplicated technical memorandum on "Some considerations relating to the effects of electric stimuli on consciousness" has now been prepared by the director of the federation, Major C. W. Hume, and is available free on request from the federation's office at 284, Regent's Park Road, London, N.3.

Emergency Bed Service: Applications and Admissions.—During the seven days ending January 5 the number of applications made by doctors to the London Emergency Bed Service for admission of patients was 1,836, of whom 84.41% were admitted. A white warning, indicating that the percentage of admissions fell to 82½, was issued on January 6.

Mr. Keith Lyle, ophthalmic surgeon to King's College Hospital, is one of the six foreign guest-lecturers invited by the Ophthalmological Society of Egypt to participate in its jubilee celebrations next month in Cairo.

COMING EVENTS

Royal Sanitary Institute.—The Health Congress of the Royal Sanitary Institute will be held in Hastings from April 28 to May 1.

Association of Surgeons.—The annual meeting of the Association of Surgeons of Great Britain and Ireland will be held in London from April 30 to May 2. The annual dinner is to be held at the Savoy Hotel on May 1, and the main subjects for discussion at the meeting are "The Management of Cancer of the Breast," "The Treatment of Perforated Peptic Ulcer," "Post-operative Strictures of the Bile Ducts," and "Crohn's Disease." A more detailed programme will be circulated later.

Anticholinesterases.—The British Pharmacological Society and the Fine Chemicals Group of the Society of Chemical Industry have arranged a symposium on anticholinesterases, which will be held at the Wellcome Research Institution, Euston Road, London, N.W.1, on March 27 and 28. There are to be four sections of the symposium, one of which will include a discussion on the effects of anticholinesterases on man. Further details may be obtained from the Hon. Secretary of the British Pharmacological Society, Physiology Department, King's College, Strand, London, W.C.2.

Physiotherapy Congress.—The First International Congress of Physiotherapy is to be held in London from September 7 to 12, under the auspices of the Chartered Society of Physiotherapy. Enrolment forms, and the preliminary programme, may be obtained from Miss M. J. Neilson, Chartered Society of Physiotherapy, Tavistock House (South), Tavistock Square, London, W.C.1.

International Hospital Congress.—The Eighth International Hospital Congress will be held in Church House, Westminster, from May 25 to 30, under the chairmanship of Dr. René Sand. The principal theme of the congress will be "Preventive Medicine as a Major Function of the Hospital and its Implications." In addition, plenary sessions will study specialized aspects of this subject. Further information may be obtained from the hon. secretary, Captain J. E. Stone, International Hospital Federation, 10, Old Jewry, London, E.C.2.

NEW ISSUES

British Heart Journal.—The next issue (Vol. 15, No. 1) will be available in a week's time. The contents include:

PROCEEDINGS OF THE EUROPEAN CONGRESS OF CARDIOLOGY.
COAGULATION TIME IN THE SELECTION OF CASES FOR ANTICOAGULANT TREATMENT. A. A. Fitzgerald Peel.
THE ROLE OF THE NERVOUS SYSTEM IN THE MAINTENANCE OF PULMONARY ARTERIAL HYPERTENSION IN HEART FAILURE. D. Halmágyi, B. Feikal, J. Iványi, T. Zsörsèr, M. Tényi, and Zs. Szücs.
THE EFFECT ON THE HUMAN THIRD HEART SOUND OF VARIATIONS IN THE RATE OF FILLING OF THE HEART. A. W. Sloan and Mary Wishart.
RHEUMATIC PERICARDITIS. G. T. Thomas, E. M. M. Besterman, and A. Hollman.
BIOPSY OF THE LEFT AURICLE IN MITRAL STENOSIS. J. B. Entecknap.
THE MECHANISM OF EFFORT SYNCOPE IN PRIMARY PULMONARY HYPERTENSION AND CYANOTIC CONGENITAL HEART DISEASE. Sheila Howarth and J. B. Lowe.
MITRAL INCOMPETENCE. Wallace Brigden and Aubrey Leatham.
THE ELECTROCARDIOGRAM DURING ANGIOCARDIOGRAPHY. Geoffrey Reynolds.
THE LUNG VOLUME IN HYPERKINETIC STATES. D. G. B. Richards, A. G. W. Whitfield, W. M. Arnott, and J. A. H. Waterhouse.
POTASSIUM AND THE CARDIOGRAPHIC CHANGES IN DIABETIC ACIDOSIS. C. B. Henderson.
THE EFFECT OF DIGITALIS ON THE NORMAL ELECTROCARDIOGRAM. M. Tiliakos.
ISCHAEMIC HEART DISEASE AND PERIPHERAL OCCLUSIVE ARTERIAL DISEASE. Lawson McDonald.

Issued quarterly; annual subscription £2 2s., single copy 12s. 6d.; obtainable from the Publishing Manager, B.M.A. House, Tavistock Square, London, W.C.1.

SOCIETIES AND LECTURES

Details in this column are necessarily brief. Further information and tickets where required (marked ●) should be obtained from the institution concerned.

Monday, January 12

MEDICAL SOCIETY OF LONDON.—8.30 p.m., discussion to be introduced by Drs. R. W. Cockshut and Wilfrid P. H. Sheldon: Minor Maladies of Children.

ROYAL EYE HOSPITAL.—5 p.m., Dr. T. H. Whittington: Non-paralytic Strabismus. Care and Treatment of the Squinting Child.

Tuesday, January 13

BRITISH POSTGRADUATE MEDICAL FEDERATION.—At London School of Hygiene and Tropical Medicine, 5.30 p.m., Professor A. Huggett, D.Sc.: Physiology of Parturition.

CHELSEA CLINICAL SOCIETY.—At South Kensington Hotel, 8.30 p.m., discussion to be opened by Lieutenant-General Sir William MacArthur: The Small Pocks and the Great Pocks.

●INSTITUTE OF DERMATOLOGY.—5.30 p.m., Dr. P. D. Samman: Reticuloses.

ROYAL COLLEGE OF PHYSICIANS OF LONDON.—5 p.m., Goulstonian Lecture by Dr D. A. K. Black: Body Fluid Depletion.

Wednesday, January 14

HARVEIAN SOCIETY OF LONDON.—8.15 p.m., annual general meeting; 9 p.m., Presidential Address by Dr. James Barnett: Links.

●INSTITUTE OF DERMATOLOGY.—5.30 p.m., Dr. H. Haber: Parapsoriasis, Poikiloderma, and Mycosis Fungoides.

INSTITUTE OF UROLOGY.—At St. Paul's Hospital, W.C., 4.30 p.m., Mr. Harland Rees: Differential Diagnosis of Frequency of Micturition.

MIDLAND MEDICAL SOCIETY.—At Selly Oak Hospital, Birmingham, 8.15 p.m., clinical meeting.

ROYAL FACULTY OF PHYSICIANS AND SURGEONS OF GLASGOW.—5 p.m., Dr. John Burns Lecture by Professor A. S. Johnstone: Hiatus Hernia and Peptic Ulceration of the Oesophagus.

Thursday, January 15

BRITISH INSTITUTE OF RADIOLOGY.—8 p.m., joint meeting with Institute of X-ray Technology. Open discussion: Technical Problems Affecting Radiology.

BRITISH POSTGRADUATE MEDICAL FEDERATION.—At London School of Hygiene and Tropical Medicine, 5.30 p.m., Professor G. F. Marrian, D.Sc., F.R.S.: Metabolism of the Adrenocortical Hormones.

EDINBURGH CLINICAL CLUB.—At B.M.A. Scottish House, 8 p.m., Dr. R. W. D. Turner: Surgical Treatment of Mitral Disease.

GLASGOW UNIVERSITY MEDICO-CHIRURGICAL SOCIETY.—At Glasgow University Union, 7.30 p.m., Presidential Address by Professor C. F. W. Illingworth

●INSTITUTE OF CHILD HEALTH.—5 p.m., Dr. J. M. Tanner: Growth and Development.

LIVERPOOL MEDICAL INSTITUTION.—8 p.m., Drs. David Johnston and Thomas Seager: General Practice—Present and Future: (*a*) Private Practice; (*b*) National Health Service Practice.

ROYAL ARMY MEDICAL COLLEGE.—5 p.m., Dr. F. M. R. Walshe, F.R.S.: Role of Trauma and Stress in Organic Nervous Affections.

ROYAL COLLEGE OF PHYSICIANS OF LONDON.—5 p.m., Goulstonian Lecture by Dr. D. A. K. Black: Body Fluid Depletion.

ROYAL COLLEGE OF SURGEONS OF ENGLAND.—5 p.m., Professor A. Sorsby: Cortisone in Ophthalmology.

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.—7.30 p.m., Dr. D. S. Bertram: Laboratory Studies on Filariasis in the Cotton Rat.

ST. ANDREWS UNIVERSITY.—At Physiology Department, Dundee, 5 p.m., Professor G. L. Brown, F.R.S.: Sympathetic Nervous System.

UNIVERSITY COLLEGE.—At Anatomy Theatre, 1.15 p.m., Mr. P. H. R. James: Recent Experiments in the Psychology of Vision.

Friday, January 16

BRITISH INSTITUTE OF RADIOLOGY.—5 p.m., meeting of medical members.

FACULTY OF RADIOLOGISTS.—At Royal College of Surgeons of England, 2.15 p.m., discussion to be opened by Dr. I. Macpherson, Dr. R. E. Steiner, Mr. J. A. Aylwin, and Mr. P. R. Allison: Mitral Disease.

●INSTITUTE OF DERMATOLOGY.—5.30 p.m., clinical demonstration by Dr. P. J. Hare: Xanthoma, Necrobiosis Lipoidica, and Granuloma Annulare.

●NATIONAL ASSOCIATION FOR MENTAL HEALTH.—At Royal College of Nursing, 2.30 p.m., A.G.M. Mr. Claud Mullins: Crime and Mental Health

OSLER CLUB OF LONDON.—At 11, Chandos Street, W., 7.45 p.m., Mr. L. T. Morton, Dr. W. Brockbank, Dr. R. Scott Stevenson, and Mr. R. C. Brock: History of British Medical Journalism.

ROYAL EYE HOSPITAL.—5.30 p.m., Mr. Howard Reed: Visual Fields.

ROYAL MEDICAL SOCIETY, Edinburgh.—8 p.m., Mr. G. C. Balleny: Homosexuality.

Saturday, January 17

BIOCHEMICAL SOCIETY.—At King's College, Strand, W.C., 10.30 a.m., scientific papers.

APPOINTMENTS

BORRIE, J., M.B.E., F.R.C.S., Lecturer in Thoracic Surgery, Otago University; Thoracic Surgeon, Dunedin Hospital, Dunedin, New Zealand.

COLBERT, IRENE, M.B., B.S., Assistant School Medical Officer, City of Newcastle-upon-Tyne.

EAST ANGLIAN REGIONAL HOSPITAL BOARD.—K. S. Preston, M.B., Ch.B., D.M.R.T., Assistant Radiotherapist, East Suffolk and Ipswich Hospital; J. C. Edwards, B.M., B.Ch., Anaesthetic Registrar, West Suffolk Hospital; J. C. Linn, M.B., B.S., D.M.R.D., Registrar in Radiology, Norfolk and Norwich Hospital.

ELLIOTT, RONALD W., M.D., M.Sc., D.P.H., Medical Officer of Health and School Medical Officer, County Borough of Bolton.

FALKNER, FRANK T., M.R.C.S., L.R.C.P., Research Fellowship and Director of Growth Studies, Centre Internationale de l'Enfance, Paris.

HOSPITAL FOR SICK CHILDREN, Great Ormond Street, London, W.C.—R. H. Wilkinson, M.D., Assistant Chemical Pathologist; J. E. Hall, M.B., Ch.B., Research Assistant in Anaesthesia; Colette L. Raulin, M.B., B.S., D.A., Senior Resident Anaesthetist; K. Rajasuriya, M.R.C.P., D.C.H., and N. T. Jaco, B.M., M.R.C.P., D.C.H., House-physicians.

LEITCH, IAN D., M.B., Ch.B., D.P.H., Medical Officer of Health and Principal School Medical Officer, South Shields County Borough.

MANCHESTER REGIONAL HOSPITAL BOARD.—The following specialist appointments are announced: T. R. Malloy, M.D., D.P.M., Consultant Child Psychiatrist, Booth Hall Hospital, Manchester, and Senior Child Psychiatrist to Manchester Child Guidance Clinic, and Lecturer in Child Psychiatry in the University of Manchester; S. Oleesky, M.D., M.R.C.P., Consultant Physician, Crumpsall Hospital, Manchester; Kathleen M. Rains, M.R.C.S., L.R.C.P., D.A., Consultant Group Anaesthetist, Lancaster and Kendal Hospitals; E. Li. Williams, M.D., Tuberculosis Physician, Oldham and Ashton Areas; H. N. Garrus, M.R.C.S., L.R.C.P., Assistant Physician in Geriatrics, Withington Hospital; J. W. Boland, M.B., B.Ch., F.R.C.S.Ed., D.M.R.T., Assistant Radiotherapist, Christie Hospital and Holt Radium Institute, Manchester.

MITCHELL, ROBERT, M.B., Ch.B., D.P.H., Medical Officer of Health and School Medical Officer, County Borough of Burton-upon-Trent.

NORTH-EAST METROPOLITAN REGIONAL HOSPITAL BOARD.—The following consultant appointments at the hospitals indicated in parentheses are announced: J. R. Dow, M.B., B.S., M.R.C.P., D.M.R.D., full- or maximum part-time Radiologist (Hackney). J. A. U. Morgan, M.B., B.S., M.R.C.P., full- or maximum part-time Pathologist (Hackney). Margaret D. W. Hamilton, M.B., Ch.B., D.A., part-time Anaesthetist (Eastern). A. J. Freese, F.R.C.S.Ed., L.D.S., part-time Dental Surgeon (North Middlesex and Tilbury and Riverside). J. Carter, M.R.C.S., L.R.C.P., L.D.S., part-time Dental Surgeon (King George).

REID, J. A. W., M.B., Ch.B., D.P.H., Medical Officer of Health, Jarrow United Districts, and Area Medical Officer, Durham County Council.

BIRTHS, MARRIAGES, AND DEATHS

BIRTHS

Anscombe.—On December 2, 1952, in London, to Maureen (formerly MacAndrew), wife of Anthony Rex Anscombe, F.R.C.S., a second son—Allen Maxwell.

Kramer.—On December 28, 1952, at Queen Charlotte's Hospital, London, W., to Elisabeth (formerly Dalley), M.R.C.S., L.R.C.P., wife of Ivor R. H. Kramer, L.D.S. R.C.S., a son.

Sears.—On December 30, 1952, at Hammersmith Hospital, London, W., to Dr. Janet (formerly Conn), wife of Dr. H. T. N. Sears, a son—Charles Alistair Newton.

DEATHS

Beaver.—On December 21, 1952, at Pathhead House, Cockburnspath, Berwickshire, Robert Atwood Beaver, O.B.E., M.D., D.P.H.

Buchanan.—On December 24, 1952, in a Glasgow hospital, Donald Campbell Buchanan, M.B., Ch.B., of 7, Albany Drive, Rutherglen, Glasgow.

Cheetham.—On December 30, 1952, at his home, 76, Scarisbrick New Road, Southport, Lancs, David Hubert Cheetham, M.B., Ch.B., aged 84.

Cock.—On December 22, 1952, at Royal Naval Hospital, Plymouth, Thomas Cock, M.R.C.S., L.R.C.P., Surgeon Captain, R.N., retired.

Cooke.—On December 24, 1952, Herbert William Cooke, M.R.C.S., L.R.C.P., of Main Road, Kerikeri Central, North Auckland, formerly of Wanganui, New Zealand, and of Bitterne, Southampton, Hants.

Dick.—On January 1, 1953, at "Aldersyde," Birstall, near Leeds, Yorks, Alexander Dick, M.C., M.B., Ch.B., D.P.H.

Ferguson.—On December 27, 1952, at Runtens Farm, Rotherwick, Basingstoke, Hants, Henry Arnot Ferguson, M.B., B.Ch., D.P.H., Colonel, R.A.M.C., retired.

Grant.—On December 19, 1952, at Kylintra, Dunbar, East Lothian, George Grant, M.B. C.M., formerly of City Road, Cardiff.

Hawkins.—On December 16, 1952, at 9, Britannia Square, Worcester, William Lawrence Hawkins, M.R.C.S., L.R.C.P., Surgeon Captain, R.N., retired.

Henriques.—On December 24, 1952, at the Miller Hospital, London, S.E., Carryl Vaughan Anthony Henriques, M.R.C.S., L.R.C.P.

Hessey.—On December 13, 1952, at the Middlesex Hospital, London, W., James Dodson Hessey, M.R.C.S., L.R.C.P., of 25, Norfolk Square, London, W., late of St. Leonards-on-Sea, Sussex.

Hickling.—On December 24, 1952, at 97, Moorside North, Fenham, Newcastle-upon-Tyne, Gertrude Hermine Geiler Hickling, M.D., D.P.H., aged 69.

Hicks.—On December 24, 1952, at 56, Friar Gate, Derby, Henry Thomas Hicks, F.R.C.S., aged 81.

Kinnear.—On December 22, 1952, at his home, Puddletown, Dorset, Forbes Kinnear, M.B., Ch.B.

Medlock.—On December 27, 1952, at his home, Hertford, Herts, Charles Harold Medlock, F.R.C.S

Outred.—On December 30, 1952, at Weybridge, Surrey, Charles Deane Outred, M.R.C.S., L.R.C.P., D.P.H., L.D.S., of Jordan Cottage, Tunbridge Wells, Kent, formerly of Gravesend, and Gloucester, aged 76.

Price.—On December 14, 1952, at Edinburgh, Edmund George Chambers Price, M.B., Ch.B., of St. Leonards, Boswall Road, Edinburgh.

Rahilly.—On December 30, 1952, John Maurice Bisdee Rahilly, O.B.E., M.B., B.S., Lieutenant-Colonel, R.A.M.C., retired, of Southborough, Tunbridge Wells, Kent.

Reid.—On December 21, 1952, at 161, Willesden Lane, Brondesbury Park, London, N.W., John Reid, M.D., D.P.H., aged 81.

Riley.—On December 27, 1952, at Urban House, Audley, Staffs, Albert Riley, M.B., Ch.B., aged 58.

Scotland.—On December 23 1952, at Heald Lawn, Stamford Road, Bowdon, Cheshire, Douglas Chiene Scotland, L.R.C.P.&S.Ed., L.R.F.P.S., aged 58.

Walker.—On December 20, 1952, at Bournemouth, Hants, Frederick Dearden Walker, M.B., Ch.B., D.T.M.

Walter.—On December 29, 1952, Richard Arthur Walter, M.R.C.S., L.R.C.P., of Wolborough Rectory, Newton Abbot, Devon, late of Frant Road, Tunbridge Wells, Kent, aged 87.

Wood.—On December 18, 1952, at Henry Lodge, Yatton, Somerset, William Vincent Wood, M.C., M.R.C.S., L.R.C.P., aged 77.

Any Questions?

Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.

Flatulence and Coronary Disease

Q.—*What is the pathogenesis of the flatulent dyspepsia preceding and accompanying coronary thrombosis?*

A.—The major cause of flatulence is air-swallowing. Patients who suffer from almost any type of dyspeptic pain may develop a habit of swallowing air to distend the stomach, with subsequent eructation which gives relief of their discomfort. This reaction ultimately becomes a sort of conditioned reflex to any type of discomfort felt in the upper abdomen or lower chest. Mild or even severe pain of myocardial infarction is often mistaken for "indigestion," as it is often felt low in the chest or in the upper epigastrium. In "conditioned" individuals the immediate reaction is to swallow air in an attempt to get relief. The flatulence which is thus induced may itself be productive of an additional type of discomfort, which can be a confusing factor in analysis of the symptoms.

Collection of Blood

Q.—*The collection of blood for the purpose of pathological investigation is a commonplace of modern medical practice. The little operation usually presents no difficulties, but occasionally it is extremely hard, and there must be few experienced practitioners who have not to confess to failure at one time or another. Will your expert please describe in detail the technique for obtaining a 5-ml. sample of blood with certainty, and at the first attempt, from a subject whose veins are invisible or very small?*

A.—Blood can be obtained from patients with invisible or very small veins in almost all cases if sufficient precautions are taken *before* the needle is actually inserted. The antecubital fossa veins are the best. A broad band is placed above the elbow with sufficient tension to obstruct venous flow but not affect the pulse. The patient is asked to open and close the hand several times and then to keep the hand firmly closed. The operator then palpates carefully and systematically the whole of the fossa for the resistance of a deep turgid vein. Even in the fattest arm and where veins are quite invisible, touch will give a lead. If the operator is not certain that he is feeling a vein, the band is released while the finger is still on the suspected vein and the turgidity will disappear. In very difficult cases 5 to 10 minutes may need to be spent before the operator is quite sure that he is feeling the vein. If there is a particular reason for the blood to be taken without congestion, at this point an assistant removes the band while the operator still has his finger on the vein, and when the congestion has passed the band is reapplied.

Once the vein has been located the skin of the fossa and the finger of the operator are cleaned with suitable antiseptic and the vein is again fixed at its lower palpable end by the operator's forefinger pressing firmly down on the vein and at the same time drawing the vein distally. The syringe needle is then held at an angle in the line of the vein and just above the fixing finger, and in one movement pushed deliberately and sharply into the vein. It is always better to push too deep than too superficially, and many operators in difficult cases deliberately attempt to transfix the vein. The plunger of the syringe is then withdrawn a little to create suction, and the syringe and needle *very slowly* withdrawn until blood appears in the syringe. Whenever possible always try to use a central rather than a lateral vein, as the former is better fixed in the tissues.

Intercourse after Syphilis

Q.—*How soon after the termination of treatment for syphilis should unprotected intercourse be allowed?*

A.—Marital intercourse without protection is, in the opinion of the writer, permissible six months after completion of treatment, provided, of course, that the serological reactions and cerebrospinal fluid are normal. However, it is probable that before many years have elapsed permission will be given even sooner. In the writer's experience the majority of patients do in fact have unprotected intercourse soon after the active lesions are healed, and there is no transmission of infection. There is danger only when there are open lesions on skin or mucous membrane. There is no evidence that semen itself contains *Spirochaeta pallida*: their presence there indicates an active syphilitic lesion in the course of the ejaculated fluid.

Muscae Volitantes

Q.—*What are the prognosis and treatment of muscae volitantes? A middle-aged man has had them for some years, but last year they began to increase in number, size, and density. Are any special examinations indicated?*

A.—People who experience muscae volitantes (awareness of floating specks in the eyes) usually display fine mobile opacities in the vitreous, especially in the anterior part of that chamber. Among middle-aged and elderly people such opacities are commonly found on routine examination. They are also commonly present in myopes, even before middle life, and are not seldom present in young people without any associated ocular abnormality. Such opacities require no special treatment, and the patient often learns instinctively to ignore them after a time. They are most likely to be obtrusive after rapid rotation of the eyes, or as a result of changed posture.

Although muscae volitantes are in a great majority of the cases not serious, it must be remembered that floating particles in the vitreous may also be a manifestation of retinal detachment, choroiditis, intraocular haemorrhage, and other lesions of the inner eye. Consequently their presence necessitates careful scrutiny with the ophthalmoscope. Treatment for any associated lesion can then be ordered, or, if no such lesion is present, the patient can be reassured.

Blood-stained Milk

Q.—*What is the significance of blood in the milk?*

A.—It is assumed that a cracked nipple has been excluded as a cause of the bleeding.

The occurrence of blood in the milk during the early days of lactation is a comparatively rare although well-recognized phenomenon. It may be only transitory, but sometimes it persists long enough to justify discontinuing breast-feeding. One or both breasts may be affected. Sometimes the blood is not fully mixed with the colostrum or milk, which suggests that the trouble is limited to certain ducts or alveoli. In other cases it is intimately mixed, and the milk appears brown. The condition has nothing to do with the character of the labour, with the mother's blood group, or with events arising during pregnancy. It is associated with the enormously increased blood supply and congestion of the breasts which is characteristic of the early puerperium, and fragility of the capillaries around some of the alveoli must be presumed.

The appearance of blood at this time might also, of course, be the first evidence of some lesion such as a duct papilloma, and if it recurs and persists after lactation has ceased this possibility should be kept in mind.

Mesenteric Adenitis in the Tropics

Q.—*In a few cases presenting with acute severe abdominal pain in a tropical area, the only finding on laparotomy has been extensive mesenteric lymphadenitis. What are the possible diagnoses? What investigations and treatment are advised?*

A.—It is probable that the lymphadenitis is secondary to an inflammatory condition of the bowel, and possible diagnoses include alimentary tuberculosis, enteritis, dysentery, appendicitis, diverticulitis, actinomycosis, balantidiasis, and schistosomiasis. Investigations should include at least three microscopical and cultural examinations of the faeces, and a biopsy of rectal mucosa for schistosome ova. A biopsy and microscopical examination of the affected nodes should also be arranged if further cases occur, as this examination would probably provide the clue to the diagnosis and hence to the correct treatment.

Vasomotor Attacks

Q.—*Three patients have complained of attacks of almost identical symptomatology which occur at times of real or imagined emotional stress. The symptoms are sudden tachycardia, fullness and pounding in the head, feeling of faintness, weakness of the legs, and tremor. Each attack lasts some minutes and may recur if the environment remains unchanged. The patients are: a male teacher, aged 57, whose attacks occur mostly when in buses or in crowded thoroughfares; a secretary, aged 42, who has attacks if she has to join queues, and also in cinemas and theatres; and a male research chemist, aged 28, whose attacks occur when attending meetings, going to interviews, or meeting strangers. Because of the distressing nature of these attacks and because the patients feel that they are going to "pass out" or that they must leave wherever they are, all three have become apprehensive of exposing themselves to situations which have previously provoked attacks. For instance, the secretary has given up going to cinemas and will often walk several miles rather than join a bus queue. I have not seen any of them during an attack, and therefore do not know the pulse rate, B.P., etc., but friends have said that they go very pale. Clinical examination between attacks discloses no abnormality, nor can I discover anything in their past histories which would appear to have started the attacks. Can you please advise on the nature of these attacks? Could they be due to paroxysmal tachycardia?*

A.—These attacks are surely vasomotor and do not suggest paroxysmal tachycardia. They almost certainly represent somatic manifestations of some psychiatric disorder, probably an anxiety state. Treatment is psychiatric. This entails more than reassurance, sedatives, and encouragement. Too much emphasis should not be laid on the attacks themselves, but an attempt should be made to grapple with the underlying psychoneurosis. Experienced physicians who are prepared to expend the necessary time and who have a reasonably good knowledge of psychiatry may achieve good results, but if a physician has any doubts about his ability to deal with such cases he should refer them to a psychiatrist. Drugs, apart from sedatives, are of little value in aborting the vasomotor turns.

Erosion of Teeth

Q.—*What is the cause of erosion at the neck of a tooth? How may it be prevented and treated?*

A.—Erosion is the name given to loss of tooth substance which usually occurs near the necks of teeth, so that shallow cavities are formed in the dentine. There is no doubt that such cavities are often caused by the abrasive action of toothbrushes and pastes. Sometimes the neck of the tooth is first exposed by some recession of the gum margin, perhaps due to parodontal disease or to changes which occur with age. When this is so no great force is required, and no unduly abrasive toothpaste: when the root is exposed it is no longer protected by the enamel, and the cementum which normally covers the root is quickly worn away. This type of injury is found more often on the left side of the mouth, since most people are right-handed, and it results from brushing the teeth in a crosswise direction.

Whilst the majority of cases of erosion can be explained in this way, there are some in which the cause is much more obscure. The inhalation of acid fumes has in the past accounted for some, and the drinking of unduly acid fluids, whether as medicine or otherwise, has been suggested for others. The inflammatory exudate from parodontal disease has also been blamed, though proof of this has not been forthcoming. Sometimes the term "erosion" is reserved for this small group of cases where the cause is hard to find.

Treatment of Ozena

Q.—*What is the best symptomatic treatment for ozena?*

A.—In cases of ozena the nose needs regular daily attention. First of all the nose is cleansed with an alkaline nasal spray. This helps to moisten any dry crusts and skin so that they can be more easily removed. Some patients find it necessary to use an alkaline douche, and, provided there is no respiratory infection about, it is usually safe. Occasionally, however, such a douche may be followed by otitis media. After this cleansing of the nose it should be sprayed with a mixture of stilboestrol-in-oil (0.5 mg. stilboestrol per ml.). If this treatment is carried out faithfully and regularly every morning, and sometimes every evening as well, the nose should nearly always be kept free of unpleasant odour and crusting. Unfortunately the treatment must be maintained, otherwise the condition is liable to relapse.

NOTES AND COMMENTS

Pain Around the Achilles Tendon.—Dr. ROY CLAYTON (Ashbourne) writes: In connexion with your expert's answer to the question on pain around the Achilles tendon ("Any Questions?" December 13, 1952, p. 1320), the following may be of interest. In February, 1948, I underwent a completely successful right-sided lumbar sympathectomy for the relief of one or other of the several difficult-to-differentiate arterial degenerative conditions. During the five winters prior to the operation I suffered, when walking, oft-repeated periods of pain and slight tenderness at a spot immediately above the insertion of the Achilles tendon. The pain, often intense and always crippling, was usually the precursor of threatened gangrene of the digits, which, however, never actually occurred. I always looked upon the pain as a manifestation of intermittent claudication, for it rapidly disappeared on resting. During the five years subsequent to the operation pain in the heel has been entirely absent. I should add that I also was a keen athlete and footballer in my younger days.

"Any Questions?" Book: Second Series.—This is now available, price 7s. 6d. (postage 6d. extra), from the Publishing Manager, B.M.A. House, Tavistock Square, London, W.C.1.

All communications with regard to editorial business should be addressed to THE EDITOR, BRITISH MEDICAL JOURNAL, B.M.A. HOUSE, TAVISTOCK SQUARE, LONDON, W.C.1. TELEPHONE: EUSTON 4499. TELEGRAMS: *Aitiology, Westcent, London.* ORIGINAL ARTICLES AND LETTERS forwarded for publication are understood to be offered to the *British Medical Journal* alone unless the contrary be stated.

Authors desiring REPRINTS should communicate with the Publishing Manager, B.M.A. House, Tavistock Square, W.C.1, on receipt of proofs. Authors overseas should indicate on MSS. if reprints are required, as proofs are not sent abroad.

ADVERTISEMENTS should be addressed to the Advertisement Manager, B.M.A. House, Tavistock Square, London, W.C.1 (hours 9 a.m. to 5 p.m.). TELEPHONE: EUSTON 4499. TELEGRAMS: *Britmedads, Westcent, London.*

MEMBERS' SUBSCRIPTIONS should be sent to the SECRETARY of the Association. TELEPHONE: EUSTON 4499. TELEGRAMS: *Medisecra, Westcent, London.*

B.M.A. SCOTTISH OFFICE: 7, Drumsheugh Gardens, Edinburgh.